DISSERTATION ON

A STUDY ON IMPACT OF MATERNAL OBESITY ON PREGNANCY OUTCOME

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations
for the award of the degree of

M.S. OBSTETRICS AND GYNAECOLOGY

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THANJAVUR - 613 004

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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APRIL -2017

CERTIFICATE

This is to certify that this dissertation entitled "**A STUDY ON IMPACT OF MATERNAL OBESITY ON PREGNANCY OUTCOME"** is a bonafide original work of **Dr.SWATHY T.M.** in partial fulfillment of the requirements for M.S Branch -VI (Obstetrics & Gynaecology) Examination of the Tamilnadu Dr.M.G.R. Medical University to be held in APRIL - 2017. The period of study was from Sept 2015 to August - 2016.

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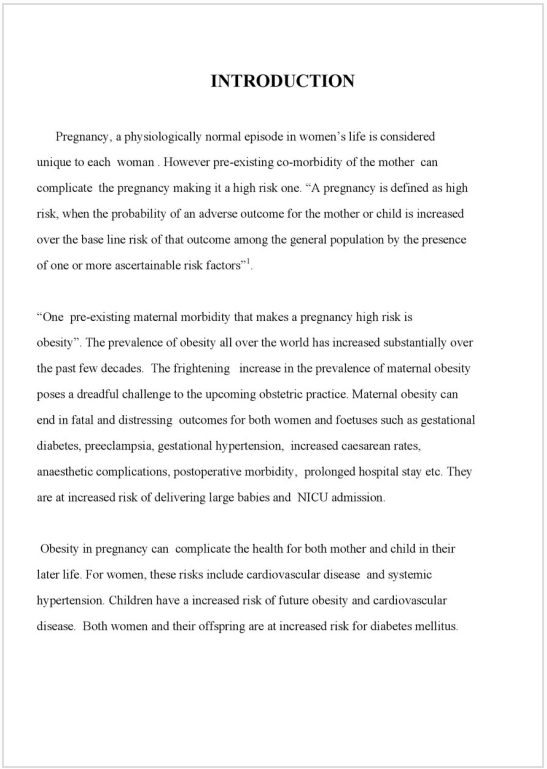
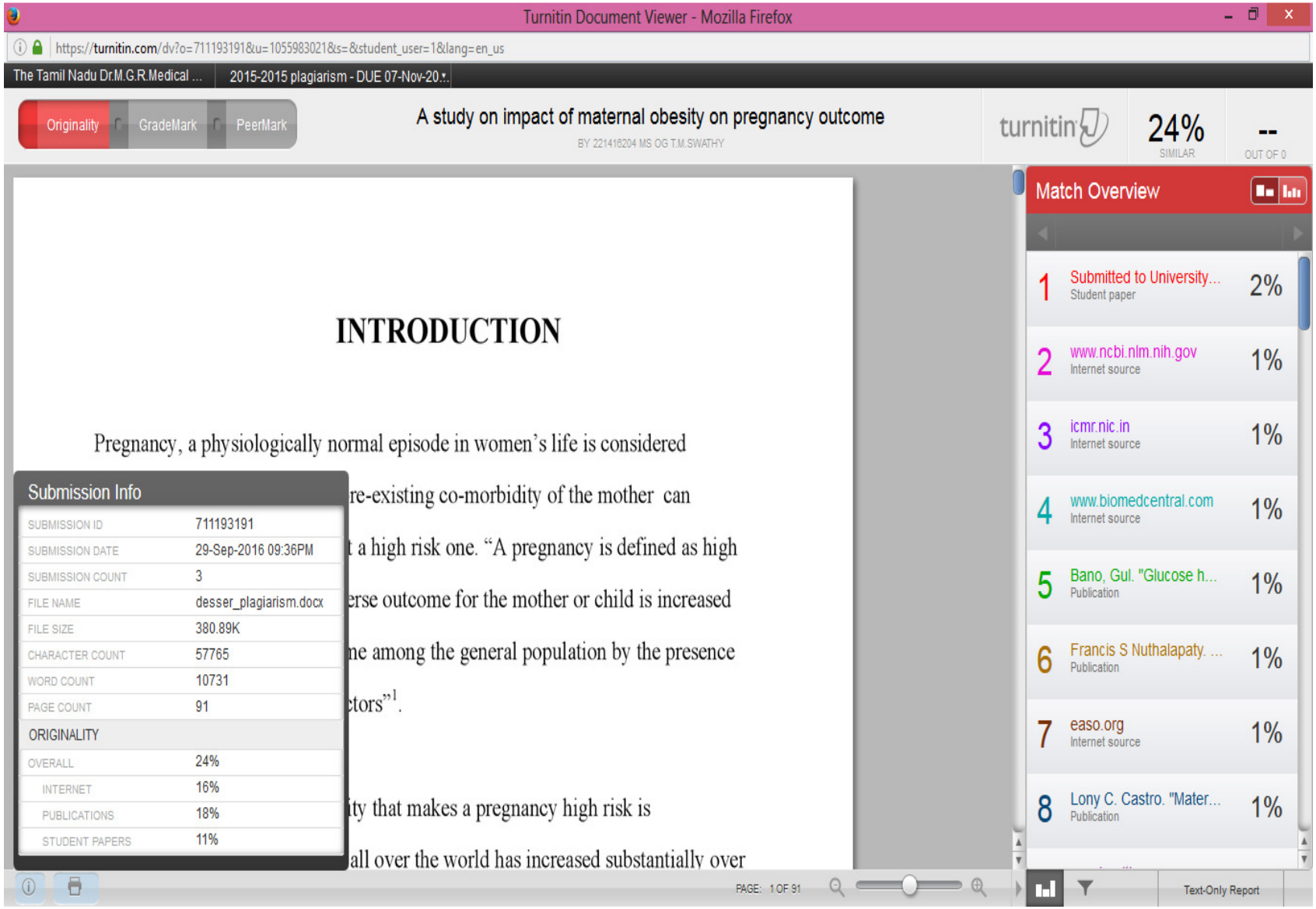


INTRODUCTION

Pregnancy, a physiologically normal episode in women's life is considered unique to each woman . However pre-existing co-morbidity of the mother can complicate the pregnancy making it a high risk one. "A pregnancy is defined as high risk, when the probability of an adverse outcome for the mother or child is increased over the base line risk of that outcome among the general population by the presence of one or more ascertainable risk factors"[1].

"One pre-existing maternal morbidity that makes a pregnancy high risk is obesity". The prevalence of obesity all over the world has increased substantially over the past few decades. The frightening increase in the prevalence of maternal obesity poses a dreadful challenge to the upcoming obstetric practice. Maternal obesity can end in fatal and distressing outcomes for both women and foetuses such as gestational diabetes, preeclampsia, gestational hypertension, increased caesarean rates, anaesthetic complications, postoperative morbidity, prolonged hospital stay etc. They are at increased risk of delivering large babies and NICU admission.

Obesity in pregnancy can complicate the health for both mother and child in their later life. For women, these risks include cardiovascular disease and systemic hypertension. Children have a increased risk of future obesity and cardiovascular disease. Both women and their offspring are at increased risk for diabetes mellitus.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DECLARATION

I, **Dr. SWATHY T.M.**, solemnly declare that dissertation titled "**A STUDY ON IMPACT OF MATERNAL OBESITY ON PREGNANCY OUTCOME"** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during September 2015 to August 2016 under the guidance and supervision of **Prof.Dr.S.PRADEEBA, M.D.,OG.,** Head of the department, Department of Obstetrics and Gynaecology, Thanjavur Medical College, Thanjavur.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University towards partial fulfillment of requirement for the award of **M.S Degree (Branch -VI) in Obstetrics and Gynaecology**.

Place: Thanjavur

Date: **(Dr. SWATHY T.M)**

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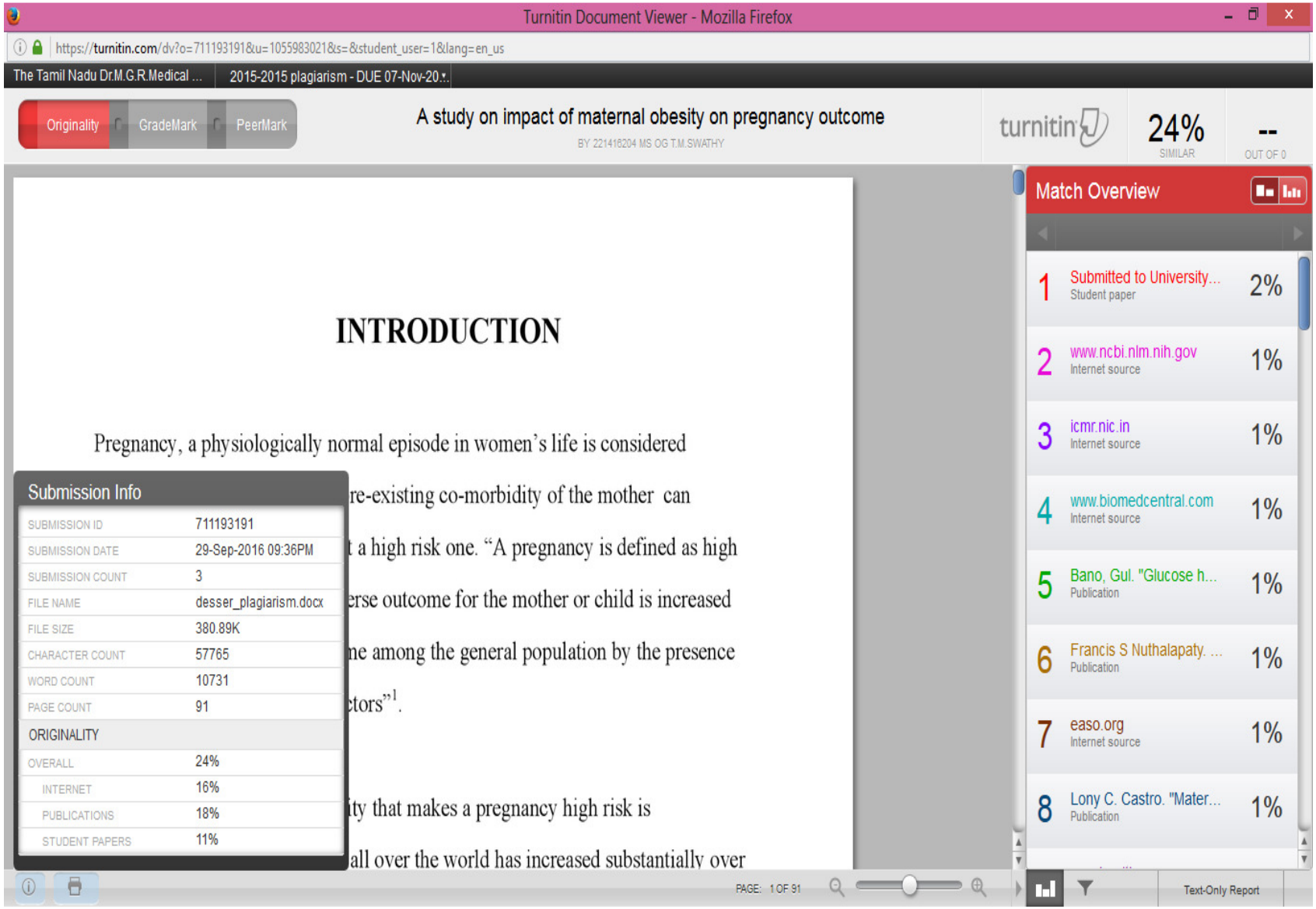
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The prevalence of obesity all over the world has increased substantially over the past few decades. The frightening increase in the prevalence of maternal obesity poses a dreadful challenge to the upcoming obstetric practice. Maternal obesity can end in fatal and distressing outcomes for both women and foetuses such as gestational diabetes, preeclampsia, gestational hypertension, increased caesarean rates, anaesthetic complications, postoperative morbidity, prolonged hospital stay etc.

They are at increased risk of delivering large babies and NICU admission. Obesity in pregnancy can complicate the health for both mother and child in their later life.

For women, these risks include cardiovascular disease and systemic hypertension. Children have a increased risk of future obesity and cardiovascular disease. Both women and their offspring are at increased risk for diabetes mellitus. Although routine weighing of pregnant women is done at each antenatal visit, not much importance is given to the weight of the women as such. In fact pre-conceptional counselling plays a vital role in women who are obese and advice on weight reduction before planning on pregnancy will go a long way in reducing the morbidity due to obesity in pregnancy to give a good maternal and fetal outcome.

AIMS AND OBJECTIVES

The aim of the study was to analyse whether obese women have an increased risk of pregnancy complications and adverse foetal outcome.

REVIEW OF LITERATURE

PREVALANCE- WORLDWIDE

The world health organisation has commented overweight and obesity as being one of the most common public health problem with an increasing prevalence in both developed and developing countries[2]. In the year 2014, 39% of adults aged 18 and above were overweight with a BMI $\geq$ 25 kg/m^2 and 13% were obese with a BMI $\geq$30 kg/m^2 of which 11% were men and 15% were women. Thus, nearly 2 billion adults all over the world are overweight and, of these, more than half a billion are obese[3].

Worldwide , women were found more likely to be obese than men. By the year 2000, 28% of men and 33% of women were obese (Ogden 2012)[4]. The global age-standardized prevalence of obesity nearly doubled from 6.4% in 1980 to 12.0% in 2008 (Gretchen et al 2012)[5].The incidence of obesity in pregnancy varies from 6% to 28% during pregnancy, depending on the obesity definition, year and characteristics of the study population[(6-9)].

PREVALENCE IN INDIA

The second most populous country, India, with a population of 1.2 billion people is experiencing a rapid epidemiological transition. Poverty causing undernutririon which had dominated in the past, is being rapidly replaced by obesity associated with wealth[10.] Industrialization and urbanization of the country has led to increased prevalence and incidence of

obesity. The Chennai Urban Rural Epidemiology Study (CURES) which was conducted in the year 2009 in Chennai city in TamilNadu had reported age standardized prevalence of generalized obesity found to be 44.9 percent, while that of abdominal obesity was 46.3 per cent. Isolated generalized obesity was found in 9.2 per cent while abdominal obesity was reported in 9.7 percent. Generalized obesity was defined as a BMI $\geq$ 25 kg/m^2 for both men and women(based on the World Health Organization Asia Pacific guidelines)[11].Abdominal obesity was defined as a waist circumference $\geq$ 90 cm for men and $\geq$ 80 cm for women[12]. Combined obesity includes both generalised and abdominal obesity.

Pradeepa et al concluded that the prevalence of abdominal obesity as well as of Generalised obesity were high in India. When extrapolated to the whole country, 134, 154 and 106 million individuals will have Generalised obesity, Abdominal obesity and Combined obesity, respectively[13].

DEFINITION OF OBESITY

"Obesity may be defined as an abnormal deposition of the adipose tissue due to hypertrophy of fat cell or hyperplasia or both which have important consequences for morbidity, disability and quality of life "[14,15].

Assessment of Obesity There are various methods[14] to assess obesity, which includes

Body Weight:

Body weight though not an accurate measure of examining fat, is a widely used index.

The various indices used are:

1. Body mass index - BMI (Quetelet's Index)

Weight in (kg)/ Height in (m2)

2. Ponderal Index

Height in (cms) Cube root of body weight in (kg)

3. Broca's Index

Height in (cm) -100

4. Lorentz's Formula

Height in (cm) -100 – Height in (cm) -150/
2 (Women) or 4 (men)

OTHER METHODS

1.Anthropometry

Skin fold thickness,

Waist circumference and Waist: hip ratio

2.Densitometry (under water weighing).

3.CT or MRI & Electrical impedance [16.]

USE OF BODY MASS INDEX (BMI)

Body Mass Index (BMI) is a simple index for measure of weight-for-height that is used to classify underweight, overweight and obesity in adults. It can be defined as the weight in kilograms divided by the square of the height in metres designated as kg/m2 [14].

According to WHO (2000) based on Body mass index (BMI) we classify as [17]

Category	**BMI range – kg/m^2**
Very severely underweight	less than 15
Severely underweight	from 15.0 to 15.9
Underweight	from 16.0 to 18.4
Normal (healthy weight)	from 18.5 to 24.9
Overweight	from 25 to 29.9
Obese Class I (Moderately obese)	from 30 to 34.9
Obese Class II (Severely obese)	from 35 to 39.9
Obese Class III (Very severely obese)	Over/equal to 40

According to Friedman and colleagues (2002) [18],

Obesity is further categorized into,

CLASS I BMI 30 to 34.9 Kg/m2 (high risk).

CLASS II BMI 35 to 39.9 Kg/m2 (very high risk).

CLASS III BMI > 40 Kg/m2 (morbid obese).

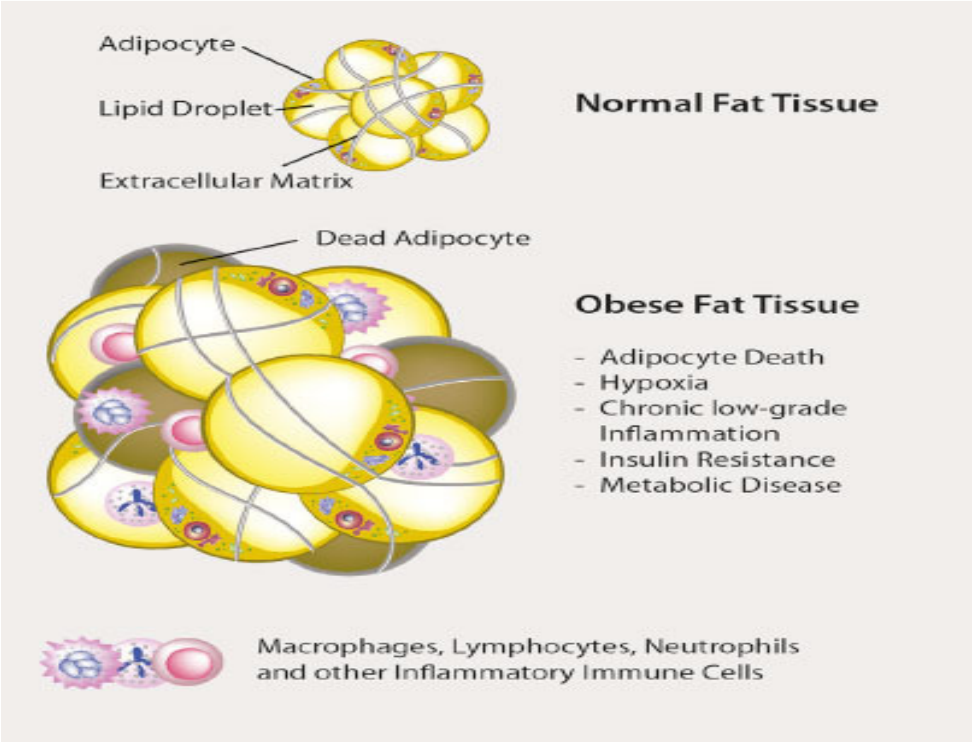
PATHOPHYSIOLOGY

Overweight and obesity arises from the imbalance between intake of food and energy expenditure, strongly influenced by genetic factors. An average human consumes one million calories per year, and an imbalance of only 1% would be enough to cause an annual weight change of 1-2 kg [16]. If the regulation is not interacting optimally with environmental conditions, even minor differences in energy intake or expenditure may lead to weight change .

In the recent days of today's world increase in sedentary lifestyle and an unlimited access to food undoubtedly has contributed to the increase in the incidence of obesity that has been increasing over time. However, the marked differences in adiposity between the individuals seem to be contributed and explained mainly by our genes [6,19]. Individuals with defects in the leptin gene[19], the pro-opiomelanocortin (POMC) Gene[20], and the melanocortin 4 receptor gene (MC4R)[21] typically present with an irresistible urge to consume food and present with early-onset obesity[16]. Adipose tissue plays an important role in the development of obesity and metabolic complications attributed to it. The two types of adipose tissue include the white and brown adipose tissue. The white adipocyte stores excess energy and offers energy to other tissues during periods of negative energy balance. When the storing capacity of the adipose tissue is exceeded or when the adipose tissue is not functioning properly, fatty acids increase in the

circulation and triglycerides accumulate in other organs such as liver, muscle, heart, and the beta cells of the pancreas[16].

FIGURE :1



Adipocyte matrix interaction play a role in the pathogenesis of obesity

ETIOLOGY OF OBESITY[16,20]

1. Genetic Factors

   Single-Gene Defects

   Polygenic Obesity

2. Environmental Factors

   Intrauterine Factors

   Early Developmental Factors

Familial and Ethnic Factors

Diet Composition and Eating Patterns

Amount of Physical Activity

Drugs

Stress and Emotional Factors

Trauma

Surgery

Infection

Endocrine and Metabolic Diseases

Abnormal Regulation of Body Weight or Body Fat

EFFECTS OF OBESITY IN WOMEN

ADOLESCENCE

PCOS

The prevalence of overweight and obesity among women suffering from PCOS is found to be high as 80%[22] . Insulin resistance resulting in hyperinsulinemia is commonly exhibited in PCOS. About one –third of obese PCOS have impaired glucose tolerance (IGT), and 7.5% to 10% have type 2 diabetes mellitus. Al Bayatti et al(2006) showed 74% patients with poly cystic ovarian syndrome had insulin resistance. Obesity augments insulin resistance and hyperandrogenism of patients with poly cystic ovarian syndrome (PCOS) and modulates β-cell function[23] .

SUBFERTILIY

Overweight and obesity is associated with increased ovulatory subfertility and anovulatory infertility[24]. Obesity has been associated with an increase in serum and follicular fluid leptin concentration and decrease in serum adiponectin levels. Leptin has been found to inhibit ovarian steroidogenesis. Lower adiponectin levels could result in increased circulating insulin. (Shilipi pandey 2010)[25]. Bellver and associates (2010) had found that the implantation, pregnancy and live birth rates were Significantly and progressively decreased with each unit of maternal BMI[26].

EFFECTS OF OBESITY IN PREGNANCY

ABORTION

Overweight and obesity can increase the risk of recurrent first trimester Miscarriage[27]. Lashen et al(2004) demonstrated the risks of early and recurrent miscarriage were significantly higher among the obese patients (odds ratios 1.2 and 3.5, 95% CI 1.01–1.46 and 1.03–12.01, respectively)[28] .

GESTATIONAL HYPERTENSION AND PRE-ECLAMPSIA

Prepregnancy maternal adiposity is the strongest modifiable risk factor for GHT and pre-eclampsia. Adipose tissue produces several inflammatory mediators which can act altering endothelial function. Interleukin 6 (IL-6) is

increased in obesity and also in preeclampsia[29]. IL-6 and TNF -alpha have been proposed as major mediators of inflammation that induces endothelial and vascular damage [(29-31)]. Obesity is complicated by oxidative stress. The origin of the oxidative stress occured secondary to increase in free fatty acids and inflammation. Obese individuals tend to have lower blood concentrations of antioxidants [32].

Sebire, (2001)[33]; Cedergren, (2004)[34] Weiss, (2004)[35] and all their colleagues found that obesity is a consistent risk factor for preeclampsia. Corrie Mcdonalds et al (2013) in her study found gestational weight gain in early pregnancy may be a potential target for interventions aimed at reducing the risk of hypertensive disorders of pregnancy[36].

GESTATIONAL DIABETES MELLITUS

Obesity is a risk factor for carbohydrate intolerance both in pregnant and non pregnant women. The fasting and post-prandial plasma insulin has been shown to be higher in obese pregnant women when compared to non obese. Weiss and associates (2004) – FASTER trail (First and second Trimester Evaluation of Risk trial) showed marked increases in gestational hypertension and diabetes in class 1 and class 2 obesity[35]. Shin et al (2010) showed the percentages of gestational diabetes attributable to overweight, obesity, and extreme obesity were 15.4%[37] .

RESPIRATORY COMPLICATION

Obesity decreases chest wall compliance and increases airway resistance and work of breathing. A decrease in forced vital capacity and forced expiratory volume at one second in noted in obese individuals during respiratory studies compared to normal weight women[38]. Sleep apnoea occurs in obesity. During pregnancy, sleep apnoea not only causes fatigue , can also increase the risk of hypertensive disorders, preeclampsia, eclampsia, and cardiovascular and respiratory disorders[38]. Further research is needed to examine the type and incidence of respiratory complications that can be associated with maternal obesity (Castro & Avina, 2002)[39].

FETAL MALPRESNTATION

Heslehurst et al. (2008) had reported significantly increased odds of malpresentation in pregnant women who are 'obese', but not in women who were 'overweight'[40].

LABOUR

Maternal obesity has a direct control and influence on mode of delivery and postnatal morbidity. The rate of induction of labour is found to be twice for obese pregnant women, compared to non-obese pregnant women. Delay in the first stage of labour is appreciably more common in obese pregnant woman, with the risk ranging from 1.5 times to 3 times more likely. Bogaerts et al (2013)[41] showed that obesity resulted in an increased incidence of

prolonged pregnancy and prolonged duration of first stage of labour. Crane SS et al (1997)[42], Kaiser PS et al (2001)[43] , Sheiner et al (2004)[44] , Dempsey et al (2005)[45] showed obese pregnant women had significant increased risk of caesarean section compared to non- obese woman. Arrowsmith et al (2011)[46] concluded that Obese women were found to have increased induction rate ending in caesarean section compared with women of normal weight following induction (37.7% versus 22.8% primiparous; 9.8% versus 7.8% multiparous women, respectively)

Seligman LC et al (2006)[47] in a study concluded obesity in prepregnant women and increased weight gain independently increase the risk of caesarean delivery, as well as of several negative outcomes with vaginal delivery. Gunilla et al (2010)[48] in a retrospective study showed overweight and obese pregnant women constitute a rapidly growing proportion of the total number of caesarean section and instrumental deliveries.

ANAESTHETIC COMPLICATION

Administering anaesthesia in a obese pregnant woman proves to be a challenge, that includes difficult placement of spinal and epidural analgesia and further complications from failed and difficult intubations (Hood 1993; Mace 2011)[49].

POSTPARTUM COMPLICATIONS

POSTPARTUM HAEMORRHAGE

Blomberg et al (2011) demonstrated an increased prevalence of postpartum haemorrhage. The risk of atonic postpartum haemorrhage(PPH) increased rapidly with increase in BMI. There was a twofold increased risk in obesity class III (1.8%)[50]. Elaine and associates (2012) in their study emphasized that primiparous obese women have a twofold increase in the risk of postpartum haemorrhage compared to women with normal body mass index (BMI) regardless of mode of birth[51]. Higher rates of postpartum haemorrhage among obese women were not attributable to their increased rates of caesarean delivery. Obesity is identified an important high risk factor for PPH.

WOUND INFECTION

The incidence of surgical wound infections is significantly related to increase in BMI (Norman , 2013)[52]. Co-morbidities such as diabetes apparently increases the risk of wound infections (Leth et al 2011)[53]. Vermillion and co- workers (2000) demonstrated that the thickness and depth of the subcutaneous tissue appears to be the only significant risk factor associated with abdominal wound infection after caesarean delivery[54]. Subcutaneous closure resulted in a modest but a significantly 6% decrease in wound disruption[55].

LACTATION

Li et al (2003)[56] and Jevitt et al (2007)[57] reported mothers who are obese (with a BMI >30) are less likely to initiate lactation, have delayed lactogenesis II, and are prone to early cessation of breastfeeding[51].

THROMBOEMBOLISM

Pregnancy as such is a hypercoagulable condition, causing venous stasis and activation of the clotting system making the women defenceless to thromboembolic events. Increased BMI can qualitatively place a pregnant obese woman in a "moderate risk" group in terms of thromboembolic events, especially if she has undergone caesarean section[58]. Despite a lack of proven evidence, the prevalence and incidence of obesity and its associated risk of venous thromboembolism warrants a careful consideration for the use of thromboprophylaxis in the obese pregnant population.

POSTPARTUM DEPRESSION

Obesity and depression have a bidirectional relationship, with each being a causal factor for the other. Lacoursiere and Varner (2009) reported that obese women had an increased incidence of postpartum depression which was direcly related to the degree of obesity CLASS I (22.6%), CLASS II (32.4%) and CLASS III (40%)[59].

CONTRACEPTION

Oral contraceptive failure is more likely to occur in overweight women. According to Holt and Colleagues (2002) women in the highest weight quartile had sixteen- fold increased risk of pregnancy[60]. Women who used very low dose OCP had 4-5 fold increase in pregnancy rate[55].

PERINATAL OUTCOME

PRETERM BIRTH

Cnattingius et al(1998)[61] found an overall increased risk for preterm birth less than 32 weeks in nulliparous with a body mass index more than 30kg/m^2, but this risk of preterm labour was no longer considerable when women with gestational hypertension and preeclampsia were excluded. Similarly, in a large population-based prospective cohort study from England, Sebire et al(2001)[62] reported there was no association between body mass index and preterm birth less than 32 weeks when analyses were adjusted for antepartum complications. The increased risk of preterm birth in obese pregnant woman was primarily associated with obesity related medical and prenatal complications and not some intrinsic predisposition to spontaneous preterm birth.

STILL BIRTH

Prevalence of stillbirths increase as the degree of obesity increases. There is 1.5 times increased risk for stillbirth in overweight and 2.1 fold risk in obese woman. Flenady and co-workers(2011) found obesity to be the the highest ranking modifiable risk factor for stiilbirth[62]. Waldenstrom,(2014) ; Yao,(2014) showed a increased incidence of otherwise mystifying late pregnancy stillbirths and early neonatal deaths to be associated with overweight and obesity[63,64].

CONGENITAL ANOMALIES

Watkins et al (2003) in the Atlantic Birth Defects Risk Factor Surveillance Study found a two to three fold increased incidence in various congenital anomalies in obese pregnant woman[65]. Rasmussen and co-workers (2008) Showed a 1.2, 1.7 and 3.1 fold increased risk for neural tube defects in overweight, obese and morbidly obese woman respectively[66]. Gilboa et al (2010) showed a correlation between body mass index and congenital heart Defects[67].

MACROSOMIA

Maternal obesity is considered a causative factor for macrosomia by mechanisms such as increased insulin resistance (even in pregnant women who do not have diabetes) resulting in higher foetal glucose and insulin

levels[68]. Placental lipases tend to metabolize the triglycerides in maternal blood, allowing free fatty acids to be transferred in excess to the growing foetus[69] .The prevalence of macrosomic newborns is increased in obese pregnant woman even when not associated with gestational diabetes as a co-morbid condition (Cedergren et al 2004)[34s .]

MORBIDITY IN CHILDREN BORN TO OBESE WOMAN

Obese woman beget obese children, who themselves become obese adults. " Barker (1992) hypothesized that adult mortality and morbidity are related to foetal and infant health, that includes both under and overgrowth". Catalano et al (2005) found a direct correlation with maternal pregestational obesity and childhood obesity[70]. They also reported significant relation with central obesity, elevated systolic blood pressure, increased insulin resistance and decreased high density lipoprotein cholesterol levels – all elements of the metabolic syndrome. Schack – Neilson et al (2005)[71], Oken (2006)[72], found a linear association of maternal weight gain with the subsequent BMI in their children.

HAZARDS OF OBESITY IN GENERAL POPULATION

An excess of body fat, and in particular of abdominal fat, is associated with multiple complications, leading to poor health.

Metabolic syndrome [73]

Refers to a collection of obesity-related metabolic abnormalities / risk factors that often co-occur in the same individuals. The essential components are

- Obesity
- Glucose intolerance
- Insulin resistance
- Lipid disturbances and
- Hypertension,

all well documented risk factors for cardiovascular disease.

Cardiovascular

Obesity can cause Coronary artery disease, stroke, heart failure, atrial fibrillation. Lavie et al states for each 1-U increase in BMI, there was an increase of 4% in the risk of ischemic stroke and 6% for haemorrhagic stroke[74].

Gastrointestinal [73]

It can result in Gastroesophageal reflux, gallbladder disease, liver steatosis and non-alcoholic steatohepatitis. Obesity-associated reflux esophagitis is thought to develop when excess abdominal fat , mechanically increases the gastric pressure and thereby reduces the frequency of esophageal sphincter relaxation with acid reflux[75].

Respiratory [16]

Obesity is a risk factor for

- Obstructive sleep apnoea
- Asthma
- Pulmonary embolism.

Bone and joints

Osteoarthritis, gout, back pain.

Dermatological

Striae, Acanthosis nigricans, Hirsutism and Fungal infection.

Cancer

Malignancies in the breast, endometrium, colon and rectum, oesophagus, pancreas, kidney, thyroid gland, gallbladder and hematopoietic system and malignant melanoma.

MANAGEMENT OF OBESITY IN PREGNANCY

DIETARY INTERVENTION

Weight reduction is not advisable in pregnancy. Recommended weight gain in obese women is 5 to 9 kilograms.

The Institute of Medicine (2009) recommended total gestational weight gain ranges for pregnant women by the prepregnancy BMI[55].

CATEGORY	BODY MASS INDEX	KILOGRAMS	POUNDS
Underweight	<18.5 kg/m2	12.5 to 18	28 to 40
Normal	18.5 to 24.9 kg/m2	11.5 t0 16	25 to 35
Overweight	25 to 29.9kg/m2	7 to 11.5	15 to 25
Obese	>30kg/m2	5 to 9.1	11 to 20

Cogswell et al (2006) reviewed gestational weight gain across BMI categories from 1990 through 2003. They reported that one third of pregnant women gained weight within the IOM recommendation[76].

PRENATAL CARE

- Universal screening for gestational diabetes and close antenatal surveillance detects most of the early signs of diabetes and hypertension.
- Screening for congenital anomalies of the foetus at 11 – 13 weeks and at 18- 22 weeks.
- Serial ultrasonography to assess the foetal growth.
- Antepartum and intrapartum foetal heart rate monitoring.
- Consider caesarean delivery if the estimated foetal weight is 5kg in obese woman without diabetes or more than 4.5 kg in an obese woman with diabetes to avoid morbidities to both the mother and foetus.

- Evaluation by the anaesthesia team at a prenatal visit or on arrival at the labour unit.
- Prophylactic antibiotics and thromboembolic prophylaxis for obese woman undergoing caesarean delivery.
- Optimal placement and type of abdominal incision to allow access to the foetus with best wound closure.
- Graduated compression stockings, adequate hydration and early mobilisation to avoid detrimental effects of thromboembolism.
- Encouraging the woman to breastfeed as it enhances postpartum weight loss and decrease the likelihood of childhood obesity.
- Counsel the mother about life style modifications and physical activity to attain the optimal weight in the postpartum period

ROLE OF BARIATRIC SURGERY

The various surgical procedures include either[55]

Restrictive malabsorptive procedures

Roux- en- Y gastric bypass

Bilio -pancreatic division with duodenal switch

Restrictive procedures

Laproscopic adjustable silicone gastric banding.

Alatishe et al (2013)[77s] reported improved fertility rates and reduced risk of obstetrical complications in women following bariatric surgery when compared with morbidly obese controls. Care must be taken to look for vitamin and nutritional sufficiency in women who have undergone bariatric surgery. When indicated vitamin B12 , vitamin D, folic acid and calcium supplementation are given.

MATERIALS AND METHODS

STUDY DESIGN : Prospective cohort study

STUDY PLACE: Thanjavur Medical College and Hospital

Thanjavur

STUDY PERIOD: September 2015 to August 2016

IN CLUSION CRITERIA

1. Pregnant women with prepregnancy BMI more than and equal to 30kg/m^2 with singleton pregnancy.
2. Non obese Pregnant woman with prepregnancy BMI between 18.5 to 24.9 kg/m^2 with singleton pregnancy

EXCLUSION CRITERIA

1. Women whose BMI < 18.5kg/m^2.
2. Women with BMI between 25kg/m^2 and 29.9 kg/m^2.
3. Women who are obese already with medical complication like diabetes, hypertension and hypothyroidism.
4. Women with post caesarean pregnancy
5. Women who could not be followed until delivery.

METHOD OF STUDY

Pregnant mothers attending antenatal out- patient department at Raja Mirasudhar Hospital ,Thanjavur medical college hospital were selected based on inclusion and exclusion creiteria. Hundred non- obese pregnant women and hundred obese pregnant women were allotted to the control and study group respectively. In the study group, women were allotted according to the class of obesity

CLASS I BMI 30 to 34.9 Kg/m^2

CLASS II BMI 35 to 39.9 Kg/m^2

CLASS III BMI > 40 Kg/m^2

In all women a detailed history followed by complete general and physical examination was done. Relevant haematological, biochemical investigations, USG were done. They were followed up to delivery and postpartum until discharge and outcome studied**.**

DATA ANALYSED

Age and parity distribution in obese and control group

They were looked for development of the following

ANTENATAL PERIOD

Gestational hypertension and pre-eclampsia

Gestational diabetes mellitus

Preterm premature rupture of membranes

Preterm labour

Abruptio placenta

Placenta praevia

Foetal presentation

LABOUR

Induction

Augmentation

MODE OF DELIVERY

Labour natural

Instrumental delivery

Caesarean section

INDICATION FOR CAESAREAN

NEED FOR THROMBOPROPHYLAXIS

POSTPARTUM

Postpartum haemorrhage

Deep vein thrombosis

Postpartum wound infection and wound dehiscence

FETAL OUTCOME

Still birth and early neonatal death

Meconium aspiration

Birth weight

NICU admissions

Indication for NICU admission

DURATION OF HOSPITAL STAY

STATISTICAL ANALYSIS

Data was collected from both groups and differences between the groups were evaluated using the chi-square and student t test and statistical significance was given at a p value of less than 0.05 ($p < 0.05$)

RESULTS AND ANALYSIS

One hundered pregnant women with BMI more than and equal to30kg/m2 and one hundred pregnant women with BMI between 18.5 to 24.5 kg/m2 were selected and followed prospectively till delivery and discharge from the hospital. A total of hundred non obese pregnant women taken as the control group and hundred obese pregnant women taken as the study group were included in the study.

TABLE : 1

AGE DISTRIBUTION

AGE	CONTROL		OBESE	
	NO	%WITH IN GROUP	NO	%WITH IN GROUP
<20 Years	1	1%	3	3%
20 to25 Years	64	64%	38	38%
26 to 30 Years	33	33%	51	51%
>30 Years	2	2%	8	8%
TOTAL	100		100	

Table : 1 shows the age distribution in both control and obese group. 64% women in the control group and 38% women in the obese group belonged to the age group of 20 to 25 years, which showed no statistical significance. 51% women in obese group were in the age group of 26 to 30 years.

CHART : 1

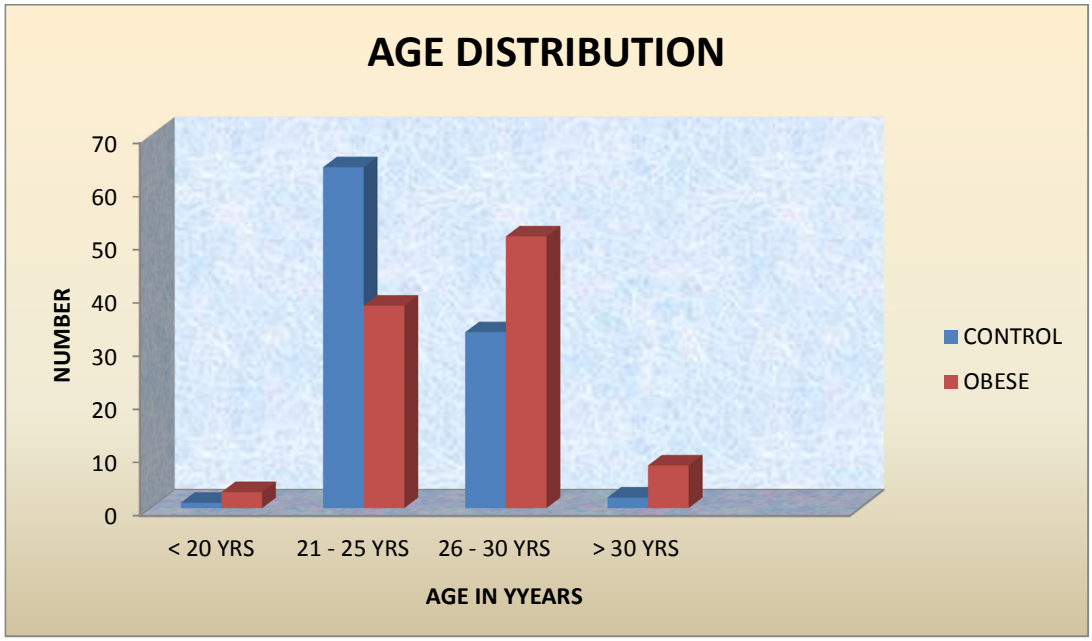


CHART : 1 :- shows 51% obese women were in the reproductive age group of 26 to 30 years

TABLE : 2

PARITY DISTRIBUTION

PARITY	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
PRIMI	57	57%	30	30%
G2 AND G3	40	40%	63	63%
G4 AND ABOVE	3	3%	7	7%
TOTAL	100		100	

Table : 2 shows parity distribution in both control and study group. 57% women in the control group and 30% women in the obese group were primi gravida. 70% women in the obese group were multigravida.

CHART : 2

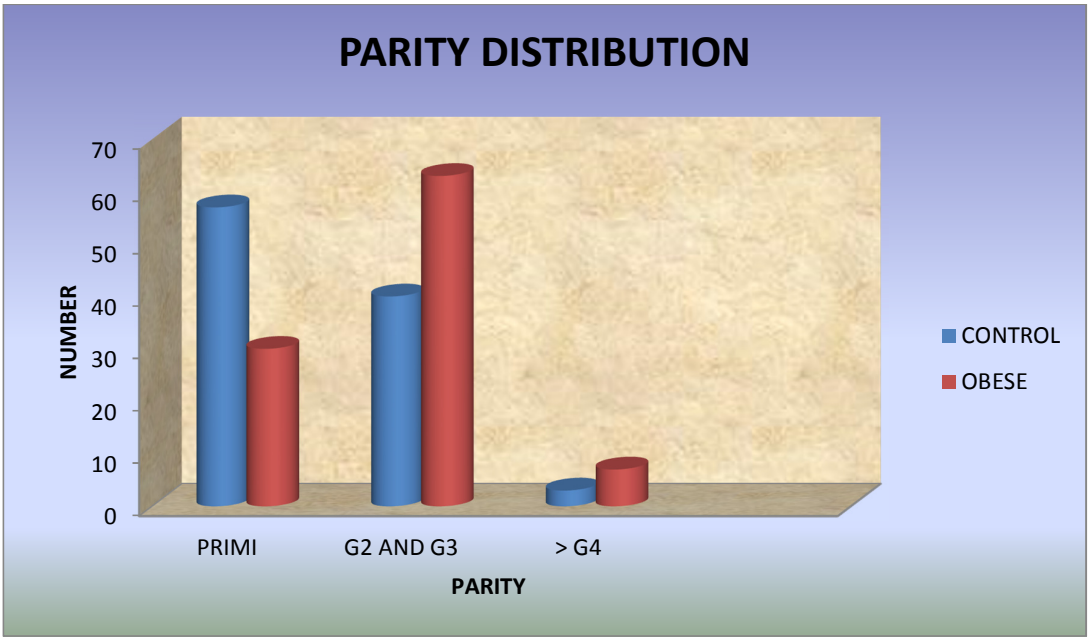


CHART 2 :- shows 70% women in the obese group were multigravidae and 30 % were primigravidae.

TABLE:3

OBESE WOMEN IN OBESITY CLASSIFICATION

CLASSIFICATION OF OBESE WOMEN	NUMBER N = 100	% WITHIN GROUP
CLASS I	72	72%
CLASS II	27	27%
CLASS III	1	1%

Table : 3 shows distribution of obese women in obesity classification. 72% women in the obese group belonged to class I and 27% women in obese group belonged to class II and 1% belonged to class III.

CHART : 3

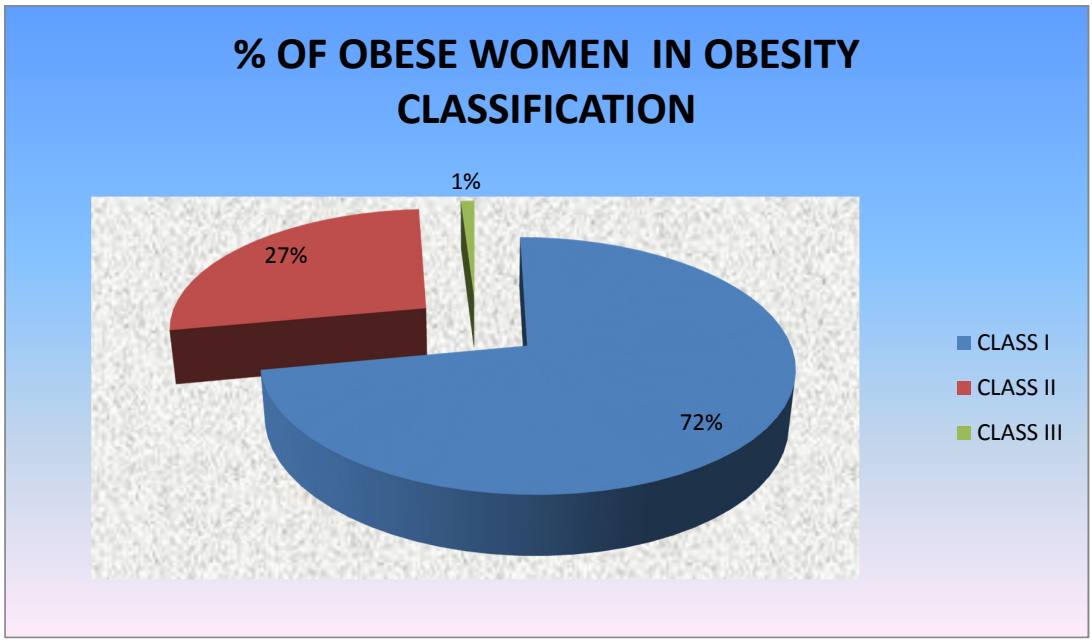


CHART 3 :- shows 72 % in obese women belonged to class II and 27% belonged to class III obesity

TABLE : 4

MEAN BMI AND WEIGHT GAIN IN CONTROL AND OBESE GROUP

MEAN BMI AND WEIGHT GAIN	CONTROL	OBESE
MEAN BMI	21.06 Kg/m2	33.70 Kg/m2
MEAN WEIGHT GAIN	10.6 Kg	9.5 Kg

Table : 4 shows the average BMI in the obese group to be 33.70kg/m2 and the average BMI in the control group to be 21.06kg/m2. The average weight gain among obese women was 9.5 kg.

CHART : 4

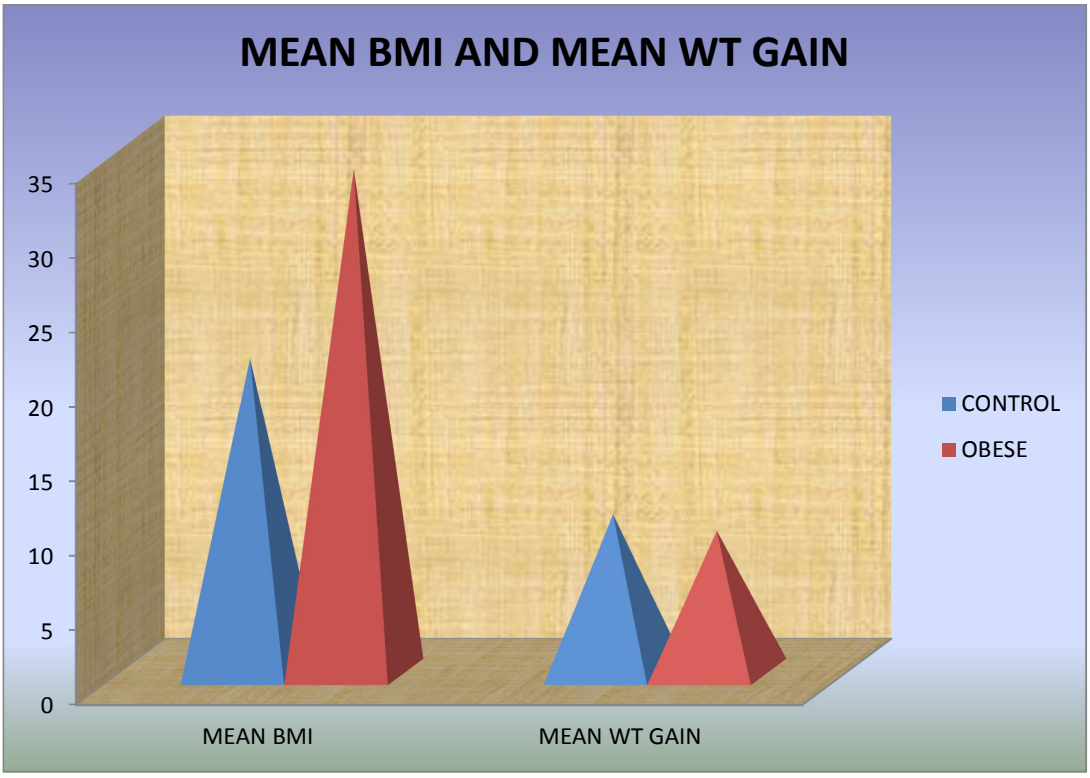


CHART 4:- shows mean BMI in the obese women was 33.7kg / m^2 and mean weight gain

TABLE : 5 GESTATIONAL DIABETES

GESTATIONAL DIABETES	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
MEAL PLAN	2	2%	4	4%
INSULIN	1	1%	9	9%

Table : 5 shows the incidence of GDM in obese group is 13% when compared to the control group with a incidence of 3%. They were found to be statistically significant with a p value <0.001.

CHART : 5

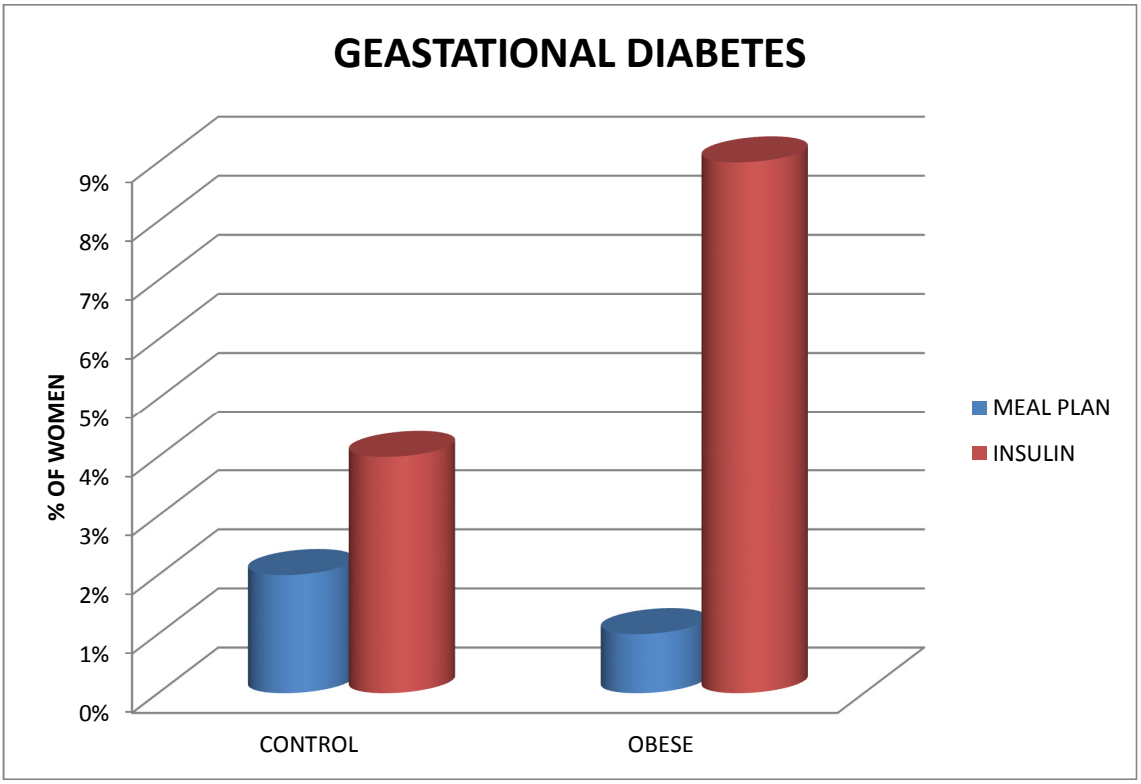


CHART 5:- shows the incidence of GDM in both obese and control group.13% women in the obese group had GDM and 3% women in control group hadGDM.

TABLE : 6

GESTATIONAL HYPERTENSION AND PRE-ECLAMPSIA

GHT ANDPRE-ECLAMPSIA	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
GHT	5	5%	28	28%
MILDPRE-ECLAMPSIA	2	2%	8	8%
SEVEREPRE-ECLAMPSIA	1	1%	5	5%

Table : 6 shows the incidence of GHT and pre- eclampsia in both control andobese group. The incidence of GHT in control group was 5% and that of pre-eclampsia was 3% whereas in obese group it was 28% for GHT and 13% forpre-eclampsia with a p value < 0.001 which was statistically very significant.

CHART : 6

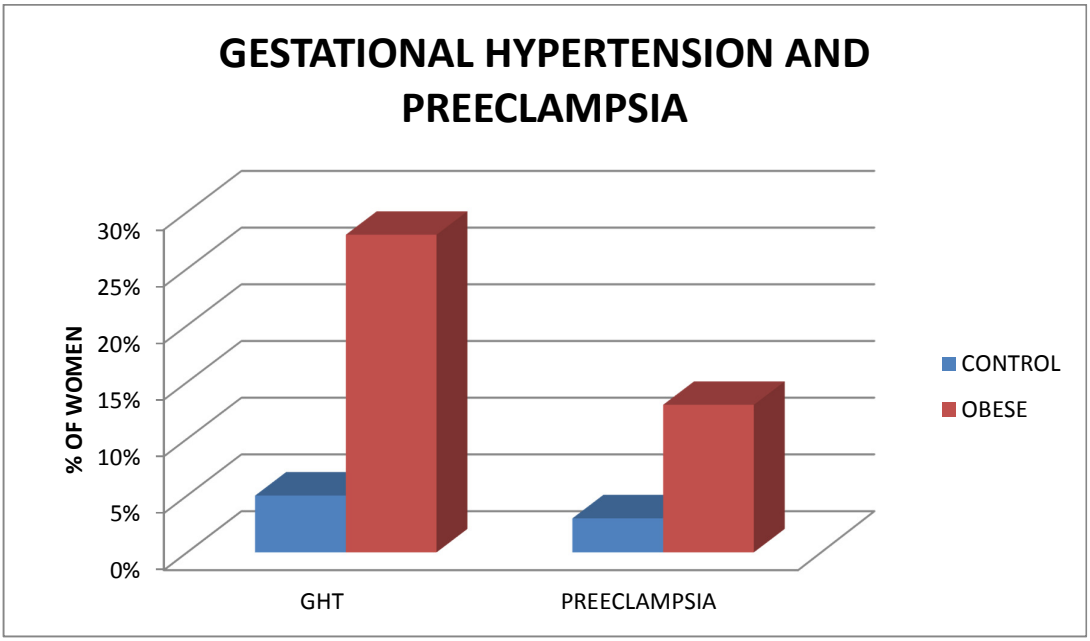


CHART 6:- shows the incidence of GHT and preeclampsia in both study and control group.

CHART : 7

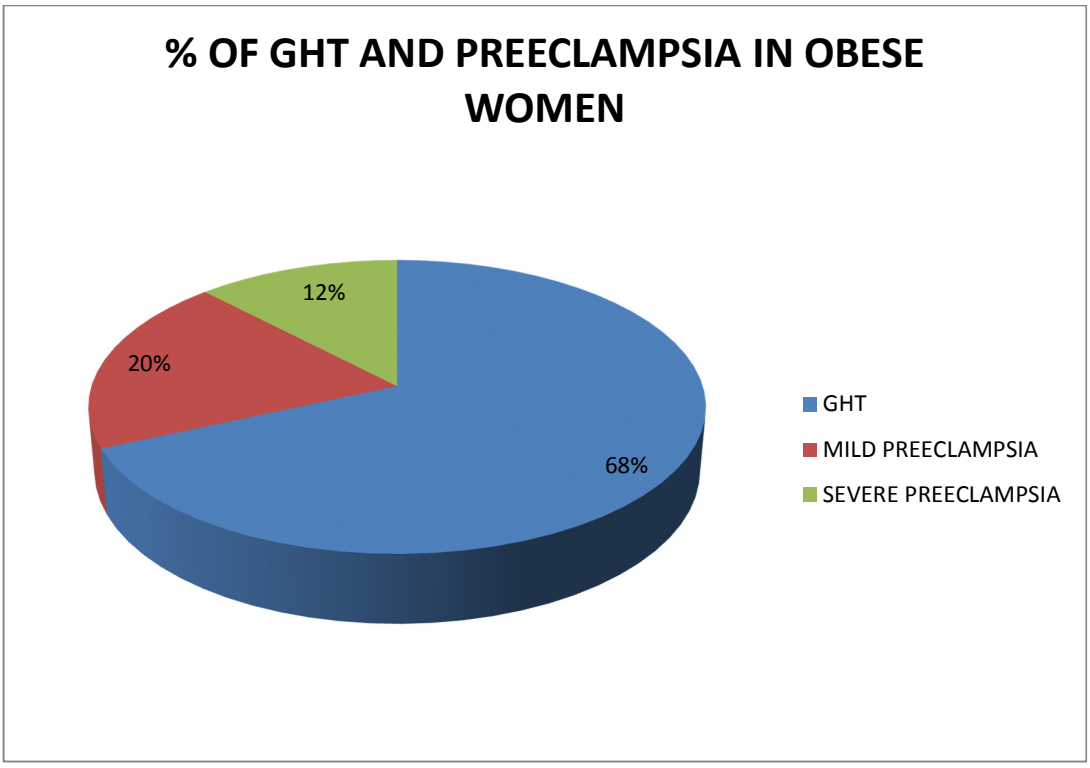


CHART :-7 shows the incidence of GHT and preeclampsia in obese women.

TABLE : 7

ANTEPARTUM HAEMORRHAGE

ABRUPTIO PLACENTA AND PLACENTA PRAEVIA	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
Abruptio placenta	1	1%	2	2%
Placentapraevia	1	1%	1	1%
Total	2	2%	1	1%

Table : 7 shows the incidence of both abruptio placenta and placenta praevia is almost similar in both control and obese group with no statistical significance.

CHART : 8

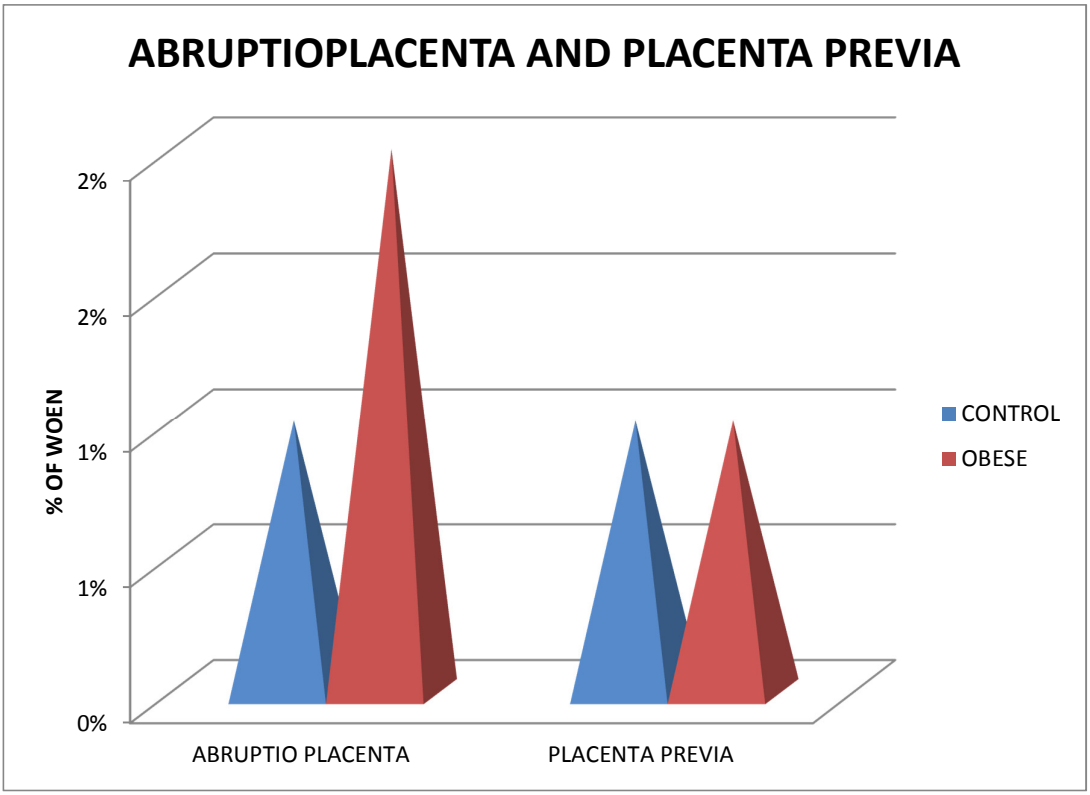


CHART 8:- shows incidence of abruption and placenta previa was similar in both groups.

TABLE : 8 PRETERM LABOUR AND PRETERM PREMATURE RUPTURE OFMEMBRANES (PPROM)

PRETERM LABOUR AND PPROM	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
PRETERM LABOUR	2	2%	1	1%
PPROM	3	3%	2	2%
TOTAL	5	5%	3	3%

Table:8 shows the incidence of preterm labour and PPROM is almost similar in both the control and obese group with no statistical significance witha p value >0.05. The incidence of preterm labour and PPROM in the obese group was 1% and 2% respectively.

CHART : 9

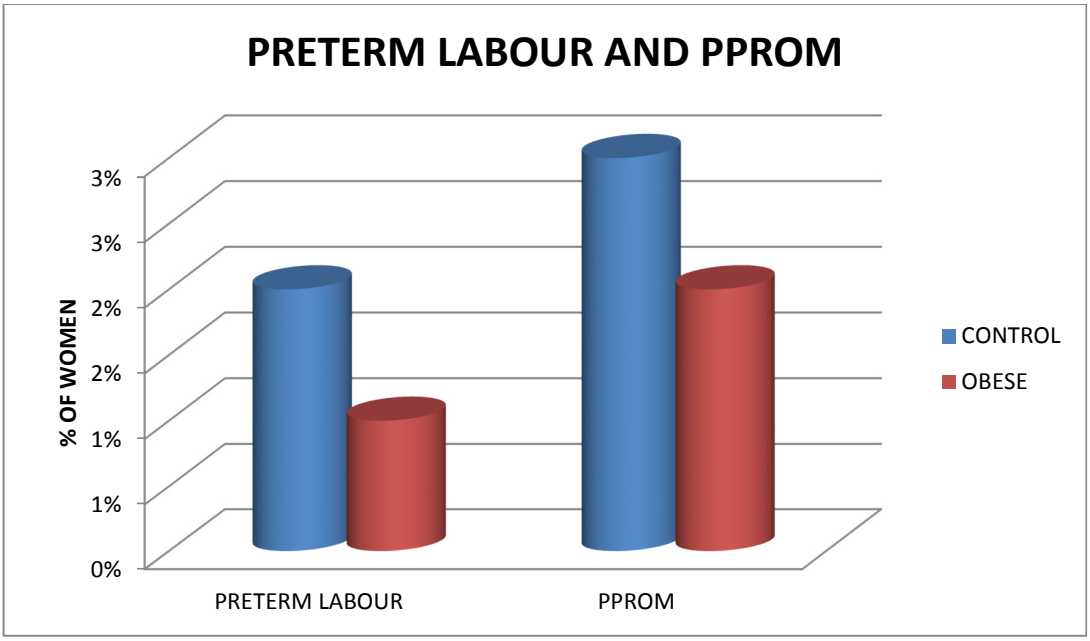


CHART 9:- shows incidence of preterm labour and PPROM was similar in both groups.

TABLE : 9

FETAL PRESENTATION

PRESENTATION	CONTROL		OBESE	
	NON=100	%WITHIN GROUP	NO N=100	%WITHIN GROUP
CEPHALIC	97	97%	96	96%
BREECH	2	2%	2	2%
TRANSVERSE	1	1%	2	2%

Table : 9 shows 96 % obese women and 97% women in the control group had cephalic presentation. The incidence of breech presentation was 2% in both control and obese group with no statistical significance.

CHART : 10

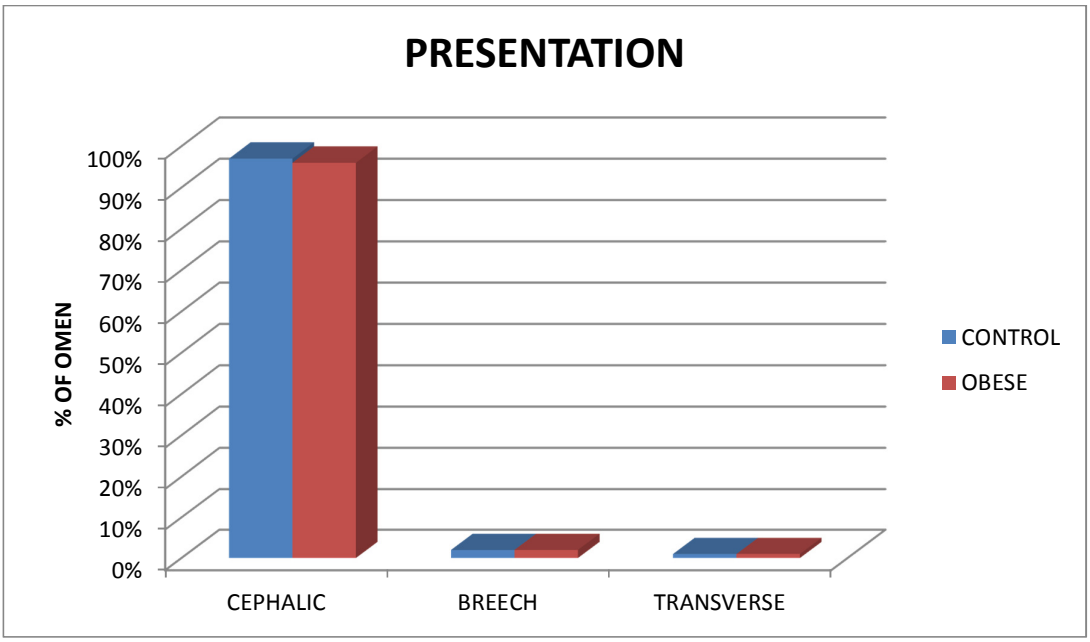


CHART 10:- depicts the incidence of cephalic presentation was 97% in control group and 96% in obese group.

TABLE : 10

INDUCTION OF LABOUR

MODE OF INDUCTION	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
PGE2 GEL INDUCTION	5	5%	14	14%
OXYTOCIN INDUCTION	6	6%	12	12%

Table : 10 shows the number and incidence of women requiring induction in both control and obese group. 26 % in obese group and 11% in control group required induction which was statistically significant with p value < 0.05.

CHART 11

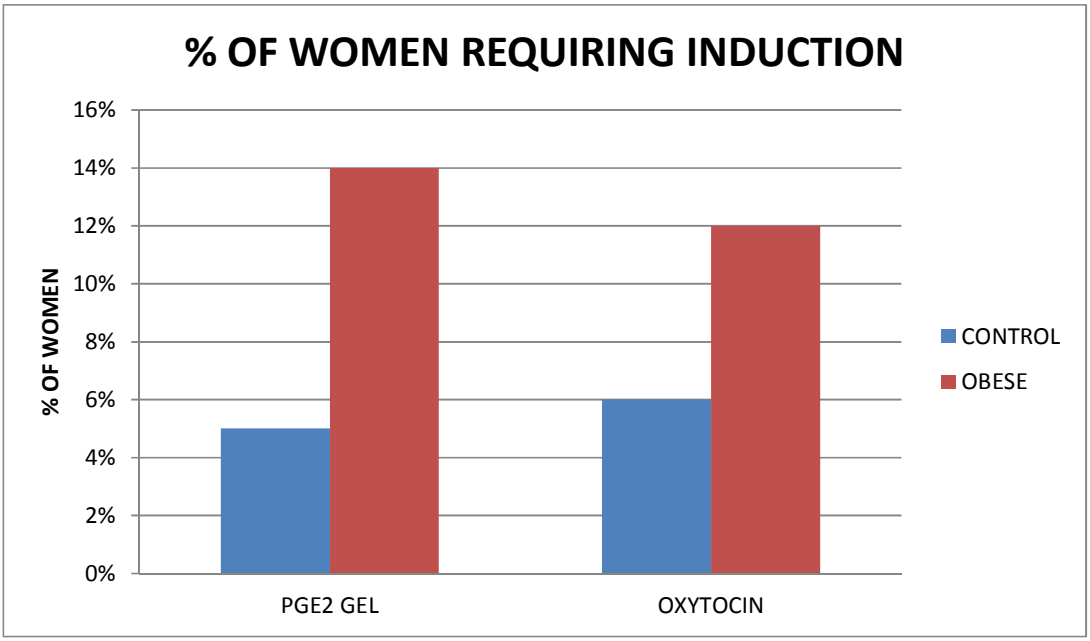


CHART 11:- 11% control and 26% in obese group required induction.

TABLE : 11

PERCENTAGE OF WOMEN REQUIRING OXYTOCIN AUGMENTATION

OXYTOCIN AUGMENTATION	CONTROL		OBESE	
	NO	% OF WOMEN	NO	% OF WOMEN
	16	16%	38	38%

TABLE 11:- shows the % of women requiring oxytocin augmentation were 38 % in the obese group and 16% in the control group with p value < 0.05 with statistical significance.

CHART : 12

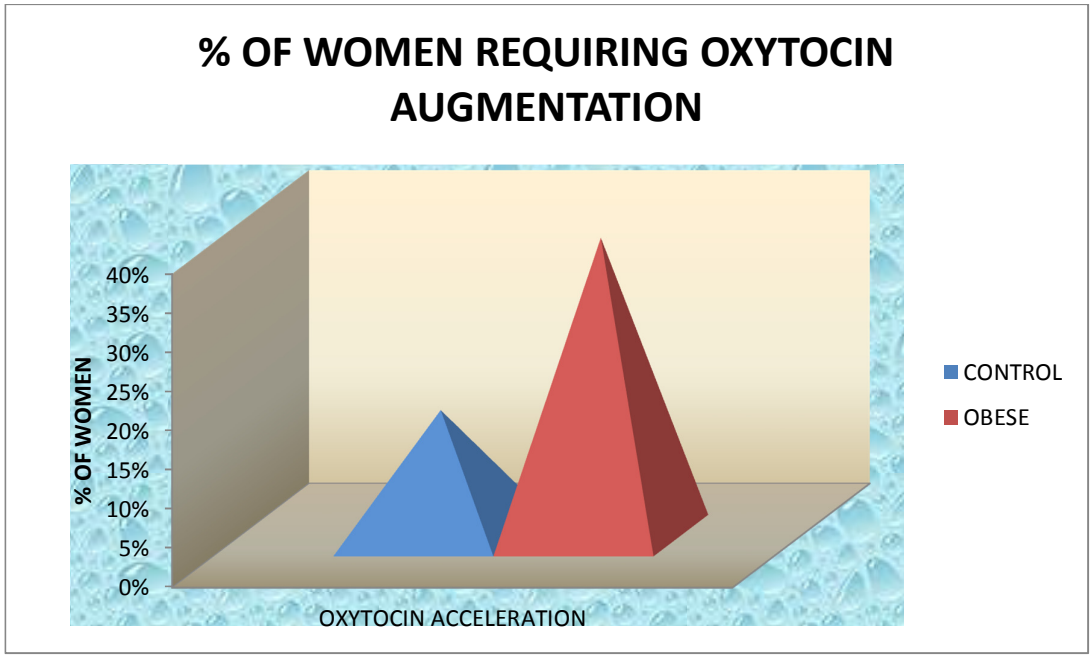


CHART :- 12 shows % of women requiring augmentation were more in obese group

TABLE : 12

MODE OF DELIVERY

MODE OF DELIVERY	CONTROL		OBESE	
	NON=100	%WITHIN GROUP	NO N=100	%WITHIN GROUP
LABOUR NATURAL	87	87%	45	45%
FORCEPS	2	2%	3	3%
VACUUM	1	1%	4	4%
CAESAREAN	10	10%	48	48%

Table : 12 shows the incidence of caesarean section was 48% in obese group and only 10% in the control group with a p value < 0.001 which was statistically very significant. 7% obese women and 3% women in control group had instrumental delivery with a p< 0.05.

CHART : 13

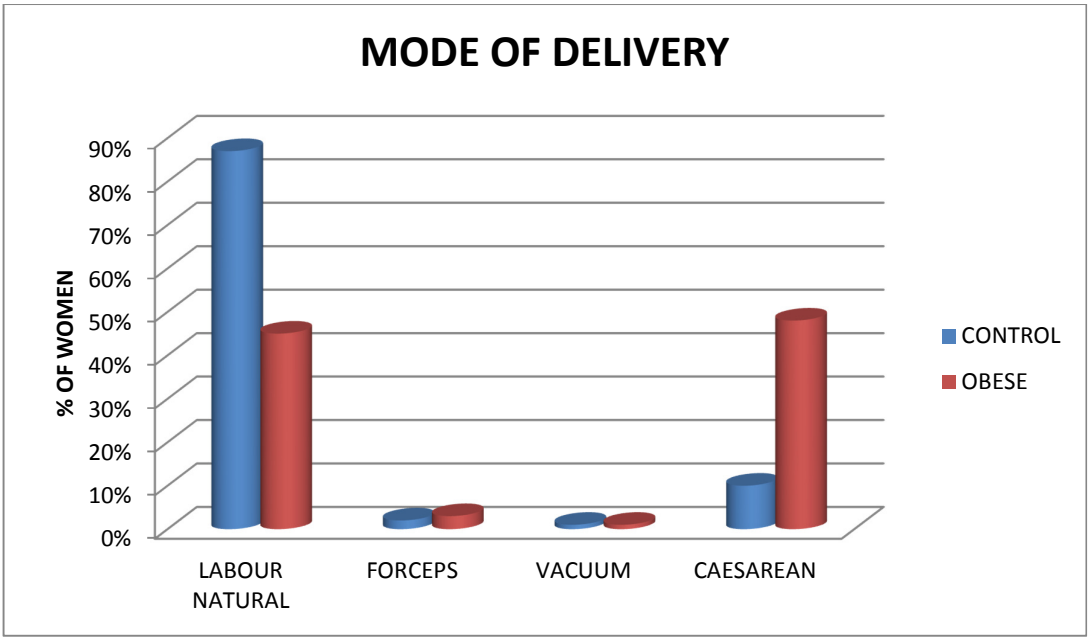


CHART :- 13 incidence of caesarean in obese group was 48 % compared to only 10% in the control group.

TABLE : 13

INCIDENCE OF CAESAREAN IN OBESE WOMEN

CLASS OF OBESITY	NO	% WITHIN GROUP
CLASS I	27	37.5%
CLASS II	20	74.07%
CLASS III	1	100%

Table : 13 shows the incidence of caesarean was 37.5 % in class I ,74.07 % in class II and 100% in class III obesity.

CHART : 14

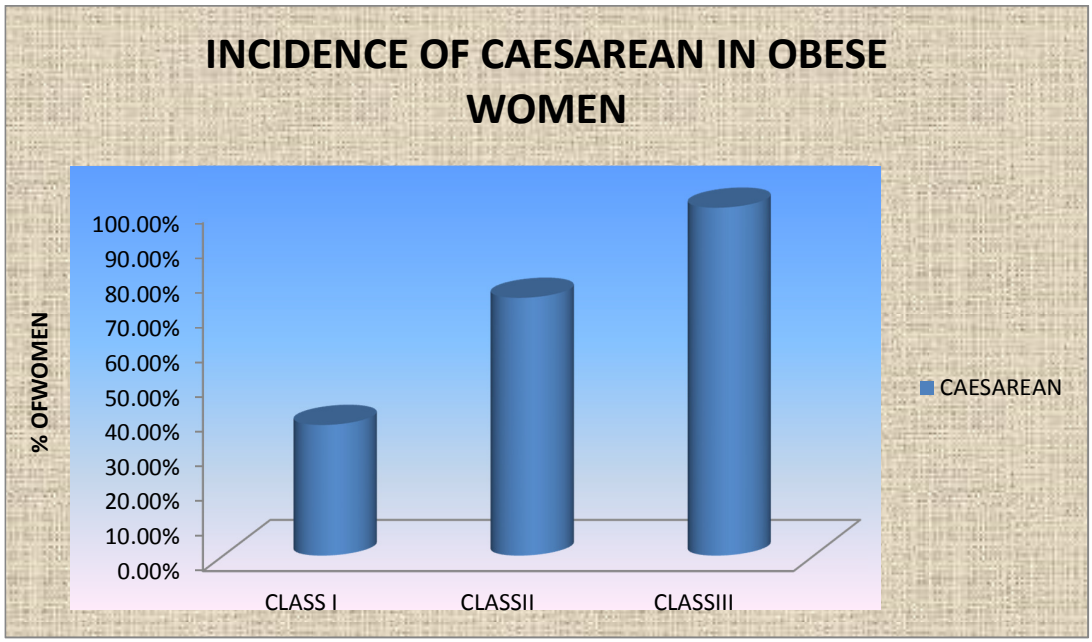


CHART 14:- shows the incidence of caesarean increased with BMI and class of Obesity.

TABLE : 14

INDICATION OF CAESAREAN

INDICATION	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
Abruption with unfavourable cervix	-	-	1	1%
Breech / foeto pelvic disproportion	2	2%	2	2%
Transverse lie	1	1%	2	2%
Cephalo pelvic Disproportion (CPD)	2	2%	15	15%
GHT with Failed induction	2	2%	12	12%
Failure to progress	-	-	8	8%
Foetal distress	1	1%	2	2%
Severe oligohydromnios	-	-	2	2%
MSAF with fetal distress	1	1%	3	3%
Placenta praevia	1	1%	1	1%

Table : 14 shows 15 % women in obese group had CPD compared to 2% women in control group. 12 % women in the obese group had failed induction as an indication for caesarean with a p value<0.001 which was statistically significant.

TABLE : 15

POSTPARTUM HAEMORRHAGE (PPH)

PPH	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
ATONIC	1	1%	1	1%
TRAUMATIC	-	-	1	1%

Table : 15 shows PPH was accounted in 1 women in the control group and 2 women in the obese group which was not statistically significant.

CHART : 15

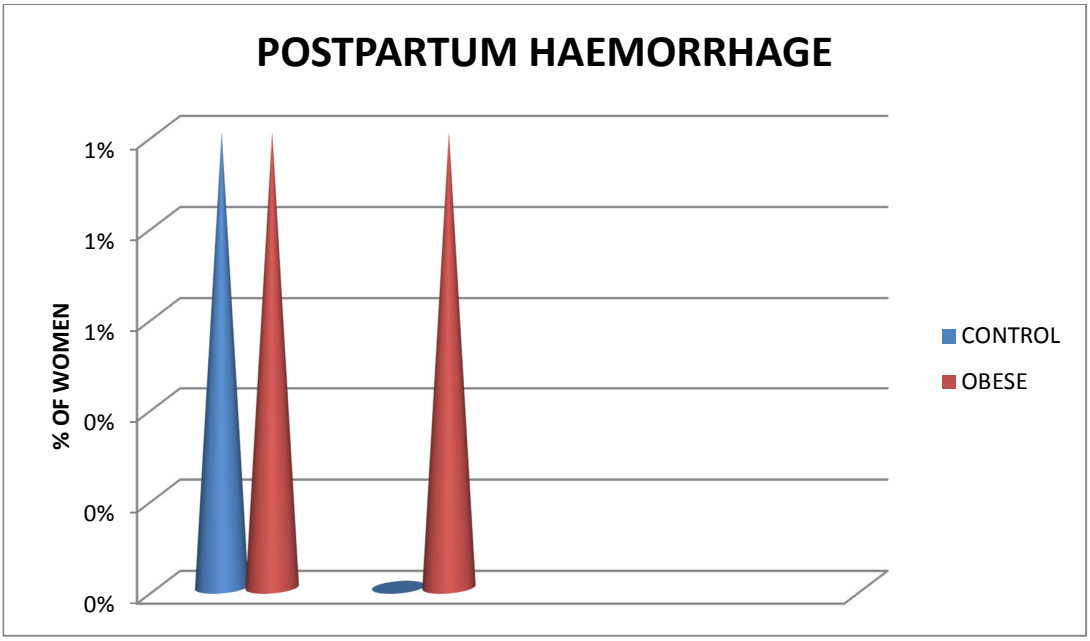


CHART 15:- shows the incidence of PPH in both the control and obese group.

TABLE : 16

POSTPARTUM COMPLICATIONS

POSTPARTUM COMPLICATIONS	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	% WITHIN GROUP
WOUND INFECTION	1	1%	8	8%
WOUND DEHISCENCE	1	1%	3	3%
DEEP VEIN THROMBOSIS	-	-	-	-

Table : 16 showed 8% and 3% incidence of wound infection and wound dehiscence respectively in the obese group. When compared with control group it was statistically significant with a p value < 0.05.

CHART : 16

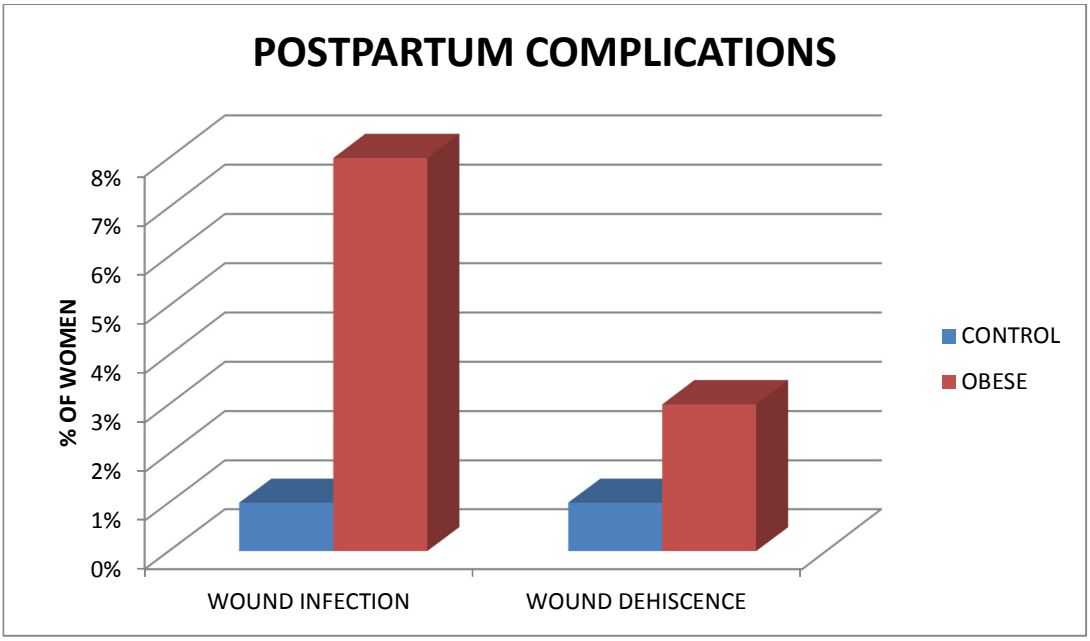


CHART 16: - shows incidence of postpartum wound infection and dehiscencewere more in the obese women.

TABLE : 17

NEED FOR THROMBOPROPHYLAXIS

NEED FOR THROMBOPHYLAXIS	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
	1	1%	48	48%

Table : 17 shows thromboprophylaxis was needed in 1% of women in the control group and 48% in the obese group with a p value of <0.001 with statistical significance.

CHART :17

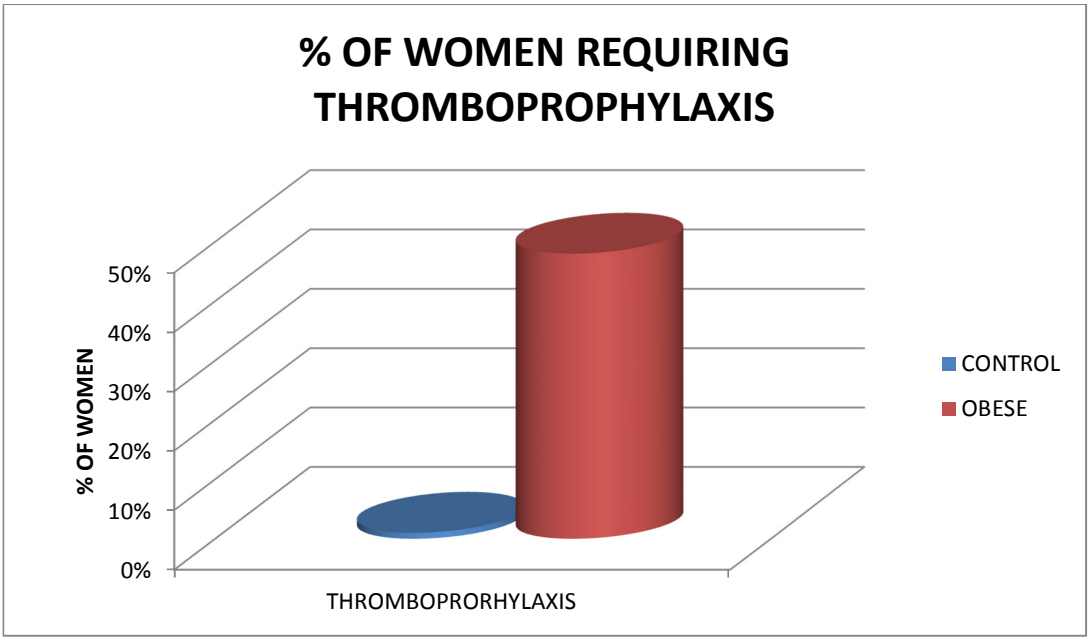


CHART 17:- shows thromboprohylaxis were required more in the obese group.

TABLE : 18

DURATION OF HOSPITAL STAY

HOSPITAL STAY		CONTROL		OBESE	
		NO	% WITHIN GROUP	NO	%WITHIN GROUP
VAGINAL DELIVERY	2 DAYS	77	77%	28	28%
	➢ 2DAYS	13	13%	24	24%
CAESAREAN DELIVERY	7 DAYS	8	8%	34	34%
	➢ 7 DAYS	2	2%	14	14%

Table : 18 showed incidence of duration of hospital stay in both obese and control group.

CHART : 18

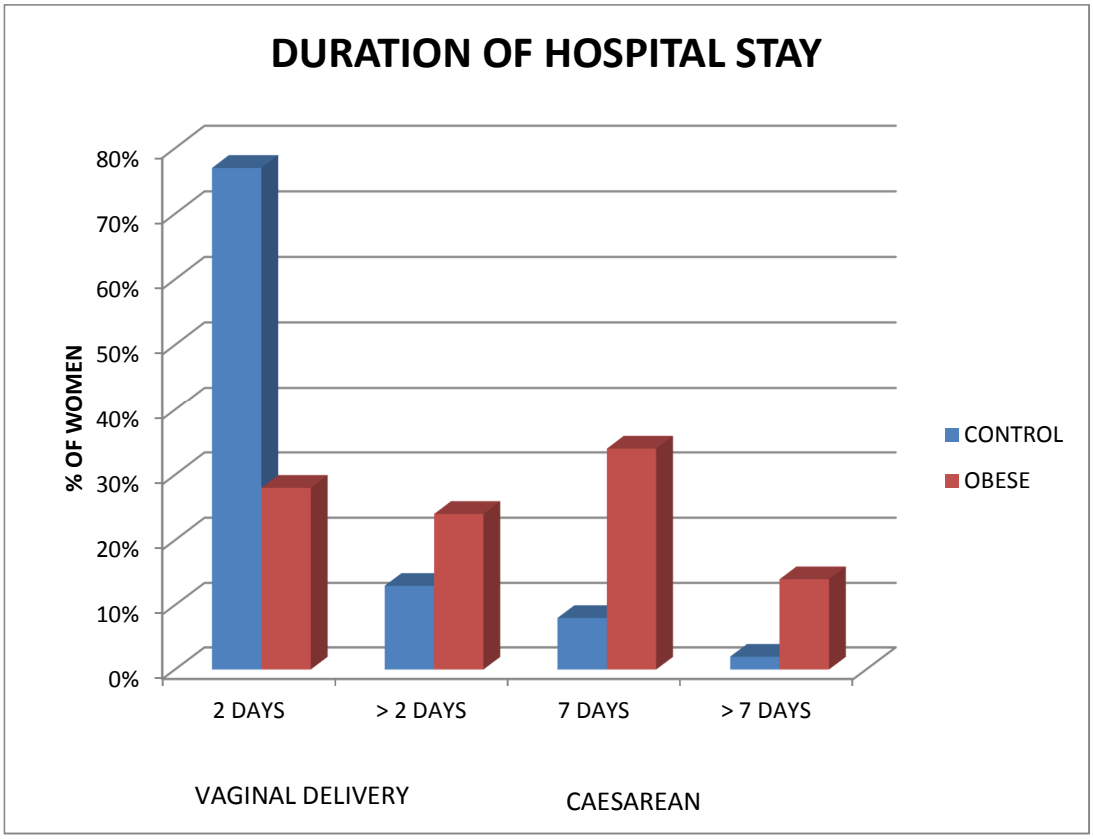


CHART 18:- shows duration of hospital stay were more in the obese women in the respective group.

TABLE : 19

AVERAGE DURATION OF HOSPITAL STAY

AVERAGEDURATION OF STAY	CONTROL	OBESE
VAGINAL DELIVERY	2.34 DAYS	3.25 DAYS
CEASAREANDELIVERY	7.2 DAYS	8.29 DAYS

TABLE : 19 shows mean duration of hospital stay in control and obese group.

TABLE : 20

FETAL OUTCOME

FETAL OUTCOME	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
LIVE BIRTH	98	98%	96	96%
DEAD BORN	1	1%	2	2%
STILL BIRTH	1	1%	2	2%

TABLE : 20 gives the incidence of live birth in both control and obese group. The incidence of live birth in obese women was 96% and in control group it was 97%.

CHART : 19

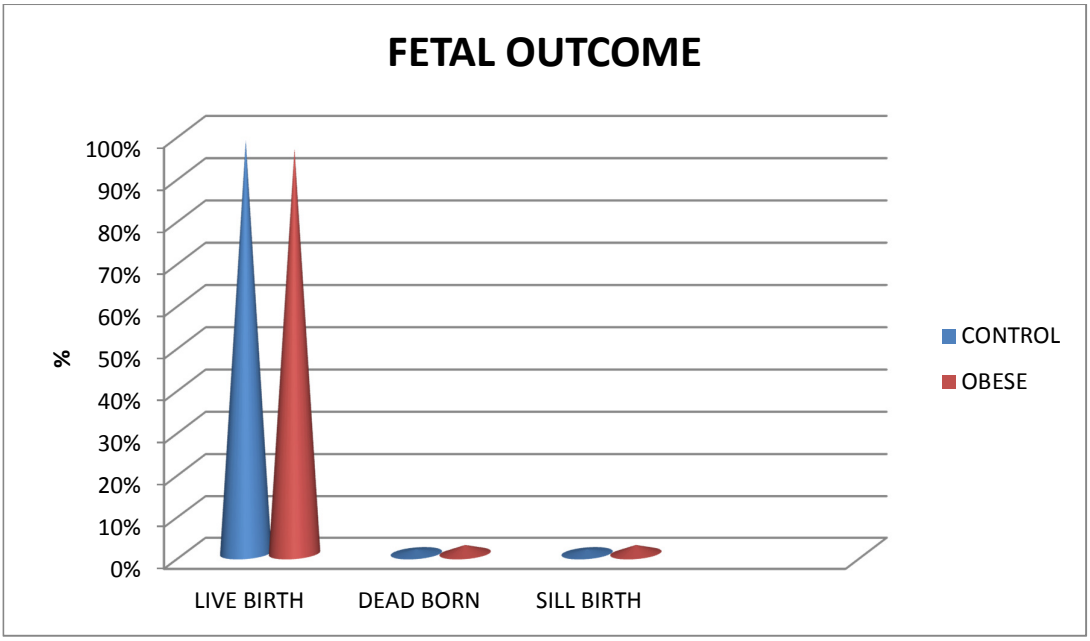


CHART 19:- shows the incidence of live birth in both groups.

TABLE : 21

BIRTH WEIGHT OF BABIES

BIRTH WEIGHT OFBABIES	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHING ROUP
Less than 2 kg	4	4%	4	4%
2 to 2.9 kg	68	68%	47	47%
3 to 3.9 kg	28	28%	46	46%
4 kg and above	-		3	3%

TABLE 21:- shows 47% babies born to obese women had birth weight 2 to 2.9kg and 46 % babies had birth weight between 3 to 3.9 kg with 3% having birthweight more than 4kg.

CHART : 20

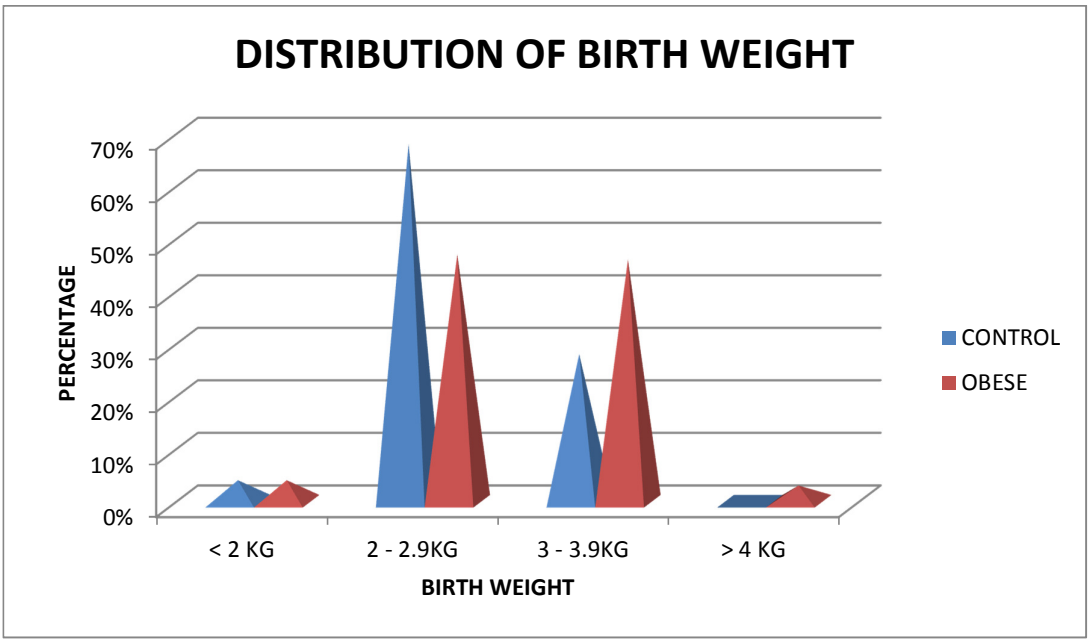


CHART 20 :- depicts the distribution of birth weight of babies.

TABLE : 22

AVERAGE BIRTH WEIGHT OF INFANTS

MEAN BIRTH WEIGHT	CONTROL	OBESE
	2.82 kg	3.21kg

Table : 22 shows the mean birth weight of infants born to obese women was 3.21 kg and in control group was 2.82kg.

TABLE : 23

STILL BIRTH

STILL BIRTH	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
	1	1%	2	2%

Table : 23 shows the incidence of stillbirth in control group was 1% and that in obese women was 2%.

CHART : 21

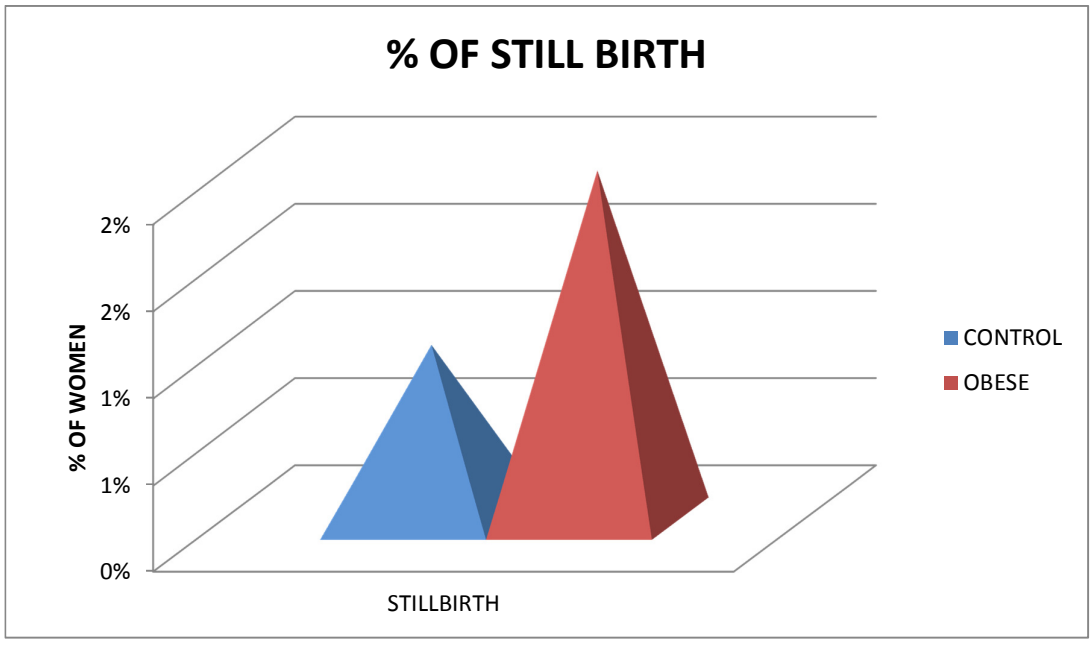


CHART 21:- shows the incidence of stillbirth in both obese and control group.

TABLE : 24

MECONIUM ASPIRATION

MECONIUM ASPIRATION	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
	1	1%	6	6%

Table:24 shows incidence of meconium aspiration was 6 % in obese women when compared to 1% in control group with a p value <0.05 which is statistically significant.

CHART :22

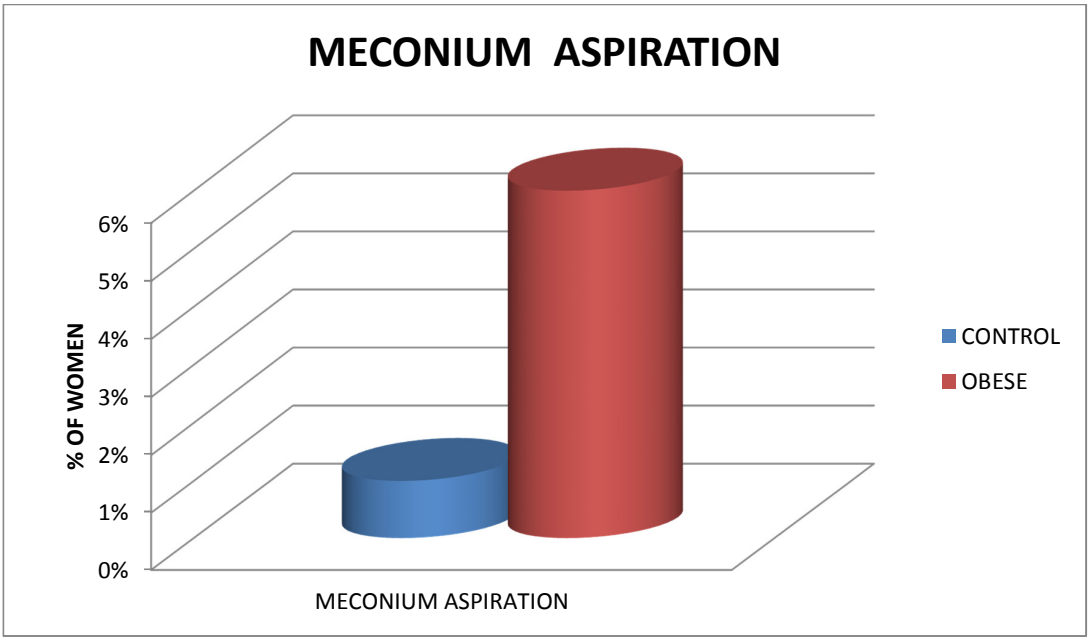


CHART 22:- shows incidence of meconium aspiration was more in obese women

TABLE : 25

NICU ADMISSIONS

NICU ADMISSIONS	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
	7	7%	22	22%

Table : 25 shows 22 % babies in obese group required NICU admissions and only 7 % babies in the control group required NICU admissions with a p value< 0.001 with statistical significance.

CHART : 23

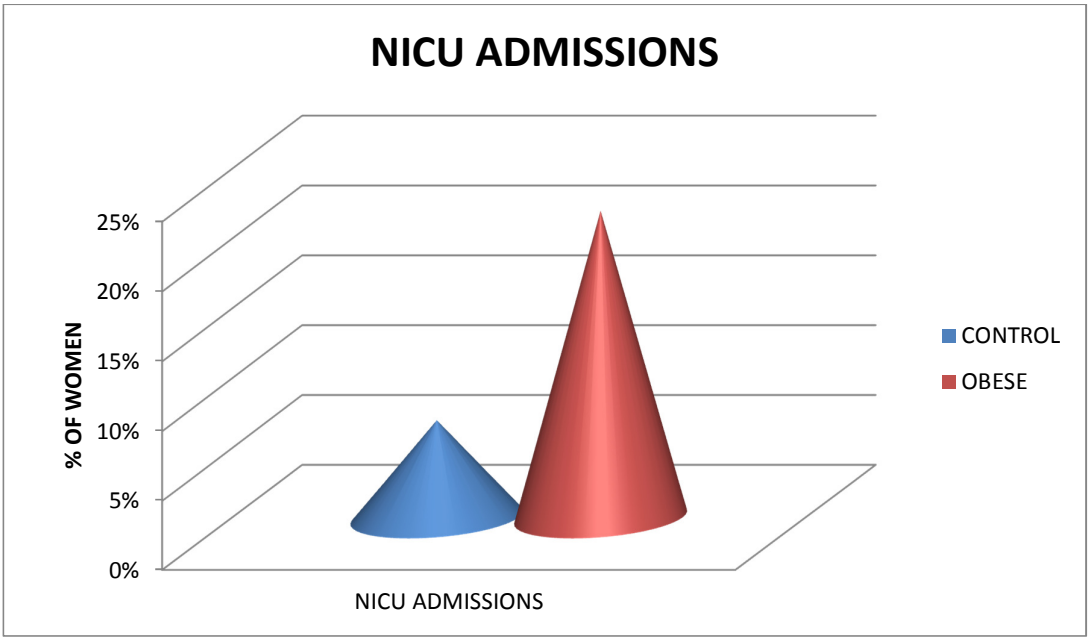


CHART 23:- shows 22% NICU admissions in the obese group and 7% in the control group

TABLE : 26

INDICATIONS FOR NICU ADMISSIONS

INDICATION	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
MECONIUMASPIRATION	1	1%	6	6%
PREMATURITY	4	4%	3	3%
BABY OF GDMMOTHER	2	2%	11	11%
MACROSOMIA	-	-	2	2%

Table : 26 shows the various reasons and the respective incidence for NICU admissions. 11% of babies in the obese group were babies born to GDM Mother. 6% of babies were admitted in view if meconium aspiration.

CHART : 24

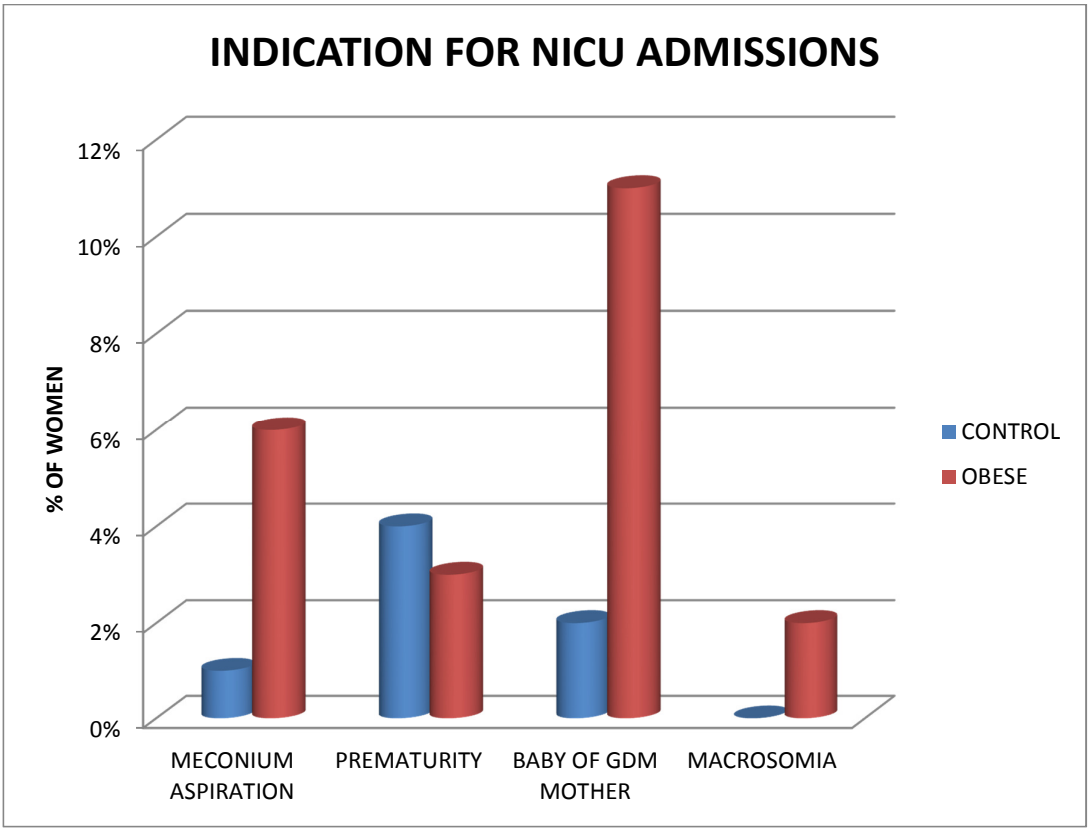


CHART 24:- shows the incidence of various causes for NICU admission

TABLE 27

EARLY NEONATAL DEATHS

EARLY NEONATAL DEATHS	CONTROL		OBESE	
	NO	%WITHIN GROUP	NO	%WITHIN GROUP
	-	-	2	2%

TABLE 27:- shows incidence of early neonatal deaths in babies born to obese women.

CHART : 25

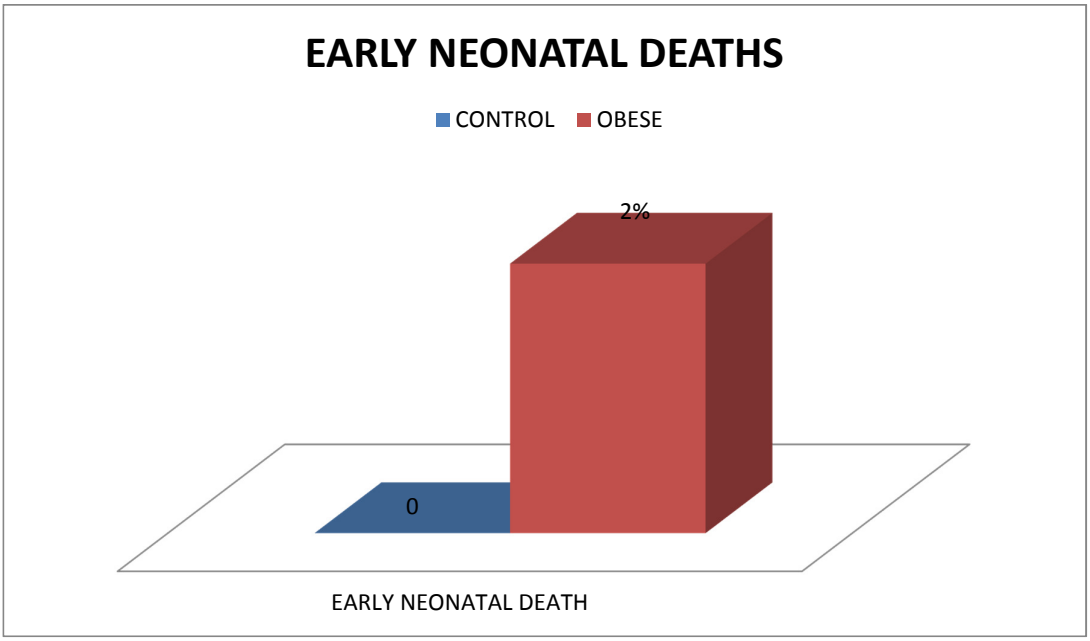


CHART 25:- shows the incidence of early neonatal deaths in the obese group.

DISCUSSION

In our study 38% women in the obese group and 64% women in the non obese group belonged to the age group of 21 to 25 years. 2% women in non obese group and 8% women in obese group were more than 30 years. 51% obese women were in the age group of 26 to 30 years and 8% were more than 30 years. This showed obese women were older than the control group women and incidence of obesity increases with age.

57 % of women in the control group were nulliparous where as in the obese group only 30 % were nulliparous. 70 % obese women were multi gravida with 7 % among them being grand multi gravida, which showed multigravida are more obese than primigravida. This is in accordance with the results of Ehrenberg HM et al (2002) that, increasing age and parity are risk factors for Obesity[8].

According to the obesity classification 72% women belonged to class I ,27% in class II and 1% women in class III obesity. Most of obese women in our study were within class I (BMI 30 to 34.9). The mean BMI in the control and obese group was 21.06 kg/m2 and 33.70 kg/m2 respectively.

The incidence of Gestational diabetes in our study was 13 % in the obese group and 3% in the control group with a significant p value < 0.001. Our results were in accordance with the Gladys et al[78] study conducted at

2005 that showed an incidence of 14 % and various other studies such as FASTER trial by Weiss and associates[35] and Shin et al (2010) showed a 15.4 % GDM attributable to overweight and obesity[37] .

In our study 48% obese women developed GHT and preeclampsia when compared to 8% in the control group with a significant p value < 0.001 that showed obesity being an important risk factor for pregnant women developing GHT and preeclampsia. This finding was consistent with O'Brien et al (2003) who showed risk of preeclampsia doubled with each 5 – 7kg/m2 increase in prepregnancy BMI[78]. Similarly Sebire, (2001)[33] , Cedergren (2004)[34] , Weiss(2004)[35] and all their colleagues found that obesity is a consistent risk factor for preeclampsia.

The incidence of abruptio placenta was 2% in obese group and 1% in the control group with no statistical significance where as Salihu et al(2009)[80] showed obese women were less likely to have placental abruption than normal weight women (adjusted odds ratio = 0.8, 95% confidence interval 0.7-0.9). The risk was similar regardless of severity of obesity.

Our study showed the incidence of preterm labour and preterm premature rupture of membranes in obese women were 1% and 2% and in the control group it was 2% and 3% respectively. Our study was not in concurrence with the study of Cnattingius et al(1998)[61] who reported an increased risk of delivery before 32 weeks in nulliparous obese women versus

lean women. (odds ratio 1.6 with a rate of 1.7% as well as a higher rate of 5-6 per 1000). Hendler et al (2005)[81] found that prepregnancy obesity was associated with fewer spontaneous preterm birth compared to women with normal body mass index which was consistent with our study.

In our study obesity was not a significant risk factor for malpresentation .The incidence of breech presentation was 2% in both the groups and the incidence of transverse lie was 2% in obese group and 1% in the control group This was not in concurrence with Heslehurst et al (2008) who showed increased odds of malpresentation in obese women[40].

The induction incidence for obese women was 26 % and that in control group was 11% which was significant. Our study showed increased induction in obese woman which were in concurrence with Arrowsmith et al (2011)[46] who concluded that Obese women had a significantly higher rate of induction of labour. 38 % women in obese group required oxytocin acceleration which was supported by Bogaerts et al (2013)[41] who showed that obesity is associated with an increased duration of pregnancy and prolonged duration of first stage of labour. The main indication for induction in the obese women were gestational hypertension, preeclampsia and gestational diabetes.

Our study showed increased caesarean section and instrumental deliveries in obese women when compared to control group. The incidence of

caesarean section was 48 % in the obese group and only 10 % in the control group. The main indications for caesarean section in the obese group was cephalopelvic disproportion (15%), failed induction (12%) and failure to progress (8%).

Our results were in concurrence with the studies if of Crane SS et al (1997) [42], Kaiser PS et al (2001)[43] , Sheiner et al (2004)[44] , Dempsey et al (2005)[45] showed obese women also have a significantly increased risk of caesarean section compared to non- obese woman.

Postpartum haemorrhage was encountered in 2 % in obese women and 1 % in control group in our study with no statistical significance which was in concurrence with Blomberg et al (2011)[50] who showed no association with maternal obesity and postpartum haemorrhage. These results was not in concurrence with Elaine and associates (2012)[51] in their study who emphasized that nulliparous obese women have a twofold increase in risk of major PPH compared to women with normal BMI. In our study the incidence of atonic PPHwas 1% in control group encountered during vaginal delivery. 2% incidence in the obese group comprised 1% atonic during labour natural and1% traumatic during an instrumental delivery.

Postpartum wound infection and wound dehiscence was increased in obese women with a incidence of 11% (8% and 3%) and 2% in the control group (1% and 1%) supported by Norman et al (2013)[52] and Vermillion et

al (2000) [54] who showed obesity to be a risk facor for postpartum wound infection and dehiscence.

The mean duration of hospital stay for obese women in case of vaginal delivery was 3.25 days and in caesarean delivery was 8.29 days and for control group was 2.34 days for vaginal delivery and 7.2 days for caesarean delivery. This was supported by Hood et al (1993) [49]and Nicole et al (2010) who showed prolonged hospital stay in obese women than in women with normal BMI.

In our study live birth incidence was 98% in control and 96% in the study group. The average birth weight of infants in the obese group was 3.21 kg and in control group was 2.82 kg. Macrosomia was accounted in 3 % babies born to obese women and none in the control group which was supported by Cedergren et al(2004)[34] who stated an increased prevalence of macrosomic newborns in obese mothers not associated with gestational diabetes.

The incidence of stillbirth was 2% in the obese women and 1% in the control group and the incidence of early neonatal death was 2% in the obese group and no neonatal death in control group ,with not much statistical significance , not supported by Sebire et al(2001)[33] and Nohr et al(2005)[82] who concluded obesity as a risk factor for stillbirth and early neonatal death. The reason could be an early intervention in obese pregnant women avoiding

a prolonged trial of labour. In our study the cause of death in the neonates in the obese group was respiratory distress and prematurity.

Incidence of NICU admissions were 22% in the obese group and 7% in the control group which showed obesity as a risk factor for increased neonatal morbidity. The main indications for NICU admissions in the obese group was meconium aspiration (6%) and infants of GDM mother (11%). The results was supported by Chen et al (2010)[83] who demonstrated a decreased apgar scores at birth in infants born to obese women.

SUMMARY

Our study was conducted at Thanjavur medical college and hospital from September 2015 to August 2016. 100 obese women with BMI more than equal to 30kg/m2 were included in the study group and 100 women with normal BMI (18.4 to 24.9 kg/m2) were included in the control group.

The following observations were made

1. 51% in obese group were in the age group of 26 to 30 years compared to 33% in the control group. Obese women tend to older .
2. 57% women in the control group and 30% women in the obese group were primigravida. 70% women in the obese group were multigravida. Obesity tend to increase with parity.
3. 72% women in the obese group belonged to class I and 27% women in obese group belonged to class II and 1% belonged to class III.
4. The average BMI in the obese group was found to be 33.70kg/m2 and the average BMI in the control group to be 21.06kg/m2.
5. The average weight gain among obese women was 9.5 kg and in the control group it was 10.6 kg.
6. The incidence of GDM in obese group was 13% and in the control group it was 3%. They were found to be statistically significant with a p value <0.001.

7. The incidence of GHT in control group was 5% and that of pre-eclampsia was 3% whereas in obese group it was 28% for GHT and 13% for pre-eclampsia with a p value < 0.001 which was statistically very significant.
8. The incidence of abruptio placenta was 1% in control and 2% in the study group and that of placenta praevia was 1% in both groups with no statistical significance.
9. Preterm labour occurred in 1% of obese group and 2% of control group.
10. PPROM occurred in 3% of control group and 2% of obese group which was not statistically significant.
11. The incidence of breech presentation was 2% in both control and obese group with no statistical significance.
12. Transverse lie was found in 1% in both study and control group.
13. 26 % women in obese group and 11% women in control group required induction which was statistically significant with p < 0.05.
14. Women requiring oxytocin augmentation were 38 % in the obese group and 16% in the control group with p value < 0.05 with statistical significance.
15. Caesarean section was done in 48% women in obese group and only 10% in the control group with a p value < 0.001 which was statistically very significant.

16. 7% obese women and 3% women in control group had instrumental delivery with a statistically significant $p < 0.05$.

17. The incidence of caesarean was 37.5 % in class I , 74.07 % in class II and 100% in class III obesity.

18. The main indication for performing caesarean section was15 % women in obese group had CPD compared to 2% women in control group and 12 % women in the obese group had failed induction for various reasons of which GHT was common p value <0.001 which was statistically significant.

19. PPH was accounted in 1 women in the control group and 2 women in the obese group which was not statistically significant.

20. 8% and 3% incidence of wound infection and wound dehiscence respectively were found in the obese group. 1% of wound infection and wound dehiscence were found in control group. When compared it was statistically significant with a p value < 0.05.

21. Thromboprophylaxis was needed in 1% of women in the control group and 48% in the obese group with a p value of < 0.001 with statistical significance.

22. The mean duration of hospital stay in and obese group was 3.25 days for vaginal delivery and 8.29 days for caesarean section.

23. The incidence of live birth in obese women was 96% and in control group it was 97%.

24. The mean birth weight of infants born to obese women was 3.21 kg and in control group was 2.82kg.

25. The incidence of stillbirth in control group was 1% and that in obese women was 2%.

26. The incidence of meconium aspiration was 6 % in obese women when compared to 1% in control group with a p value < 0.05 which is statistically significant.

27. 22 % babies in obese group required NICU admissions and only 7 % babies in the control group required NICU admissions with a p value < 0.001 with statistical significance.

28. 11% of babies in the obese group born to GDM mother and 6% of babies in obese group were admitted in view if meconium aspiration.

29. Macrosomia was recorded in 3% of babies in obese group and 2% babies required NICU admissions.

30. 2% early neonatal deaths occurred in babies born to obese mothers.

CONCLUSION

Obesity in pregnancy has numerous potential detrimental effects on the mother and baby including gestational diabetes, preeclampsia, gestational hypertension, increased induction, instrumental delivery and increased caesarean section and prolonged hospital stay. It can result in increased NICU admissions and decreased Apgar scores at birth in new born.

Obesity not only results in adverse outcomes in pregnancy, it can result in morbidity in both mother and infant in later life. It increases the incidence of type II diabetes, hypertension, cardiovascular diseases and cerebrovascular accidents in the women. Greater awareness of these adverse events needs to be made to the healthcare professionals who can target obese or overweight women of child bearing age. Such women should be informed about the risks associated with obesity and pregnancy and receive appropriate dietary and life style advice. Hence effective anti obesity strategy are needed at national level to stop this growing problem.

Finally the health and economic impact of rising obesity rates in women of reproductive years is of significant public health importance as obesity is an important modifiable risk factor for adverse pregnancy outcomes.

Further research is needed into the mechanism by which obesity adversely affects the pregnancy and how this can be modified in obese pregnant women, as obesity is proving difficult to curb and rising significantly.

This indicates the importance of pre - conceptional counselling which is the appropriate time for creating awareness regarding the hazards of obesity in pregnancy and ideal time for the interventional measures to be sought. Greater significance and awareness needs to be placed on the importance of normal weight before pregnancy.

BIBLIOGRAPHY

1. High risk Pregnancy: Management options: D.K. James
2. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser. 2000; 894():i-xii, 1-253. 37. Obesity and abdominal obesity in Asian Indians.Mohan V, Deepa R Indian J Med Res. 2006 May; 123(5):593-6.[PubMed].
3. WHO Obesity and overweight – World health organisation. www.who.int/mediacentre/factsheets/fs311/en/
4. Ogden CLet al: prevalence of obesity in the United states,2009-2010. NCHS data brief no 82, Hyattsville, National centre for healthatistics,2012.
5. Gretchen A Stevens et al ,National, regional, and global trends in adult overweight and obesity prevalences, population health metrics, Published online 2012 Nov 20. doi: 10.1186/1478-7954-10-22.
6. Abrams BF, Laros RK Jr. Prepregnancy weight, weight gain, and birthbweight. Am J Obstet Gynecol. 1986; 154;503-509.3. Abrams B, Parker J. Overweight and pregnancy.
7. Calandra C, Abell DA, Beischer NA. Masternal obesity in pregnancy. Obstet Gynecol.1981;57:8-12.
8. Ehrenberg HM, Dierker L, Milluzzi C, et al, Prevalence of maternal obesity in an urban center. Am J Obstet Gynecol. 2002; 187:1189-1193.
9. Lu GC, Rouse DJ, Dubard M, et al. The effect of the increasing prevalence of maternal obesity on perinatal morbidity. Am J Obstet Gynecol. 2001; 185:845-849.
10. Obesity and abdominal obesity in Asian Indians.Mohan V, Deepa R Indian J Med Res. 2006 May; 123(5):593-6.[PubMed].
11. Harrison GG, Buskirk ER, Lindsay Carter ER, Johnston FE, Lohman TG, Pollock ML, et al. Skinfold thickness and measurement technique. In: Lohman TG, Roche AF, Martorell R, editors. Anthropometric standardization manual.Champaign, IL: Human Kinetics Books; 1988. pp. 55–70.

12. The Asia Pacific perspective: redefining obesity and its treatment. Regional Office for the Western Pacific (WPRO), World Health Organization. International Association for the Study of Obesity and the International Obesity Task Force: St Leonards, Australia; Health Communications Australia Pty Limited. 2000:22
13. Rajendra pradeepa "Prevalence of generalized & abdominal obesity in urban & rural india- the icmr-indiab study (phase-i) [icmr-indiab-3]" indian J med res 142, august 2015, pp 139-150 dOi:10.4103/0971-5916.164234.
14. Park's Text Book of Preventive and Social Medicine.
15. Hager, A. (1981). Br.med. Bull, 37 (3) 287.
16. Pathogenesis of Obesity and Effects of Treatment "Clinical and Molecular Studies on Body Fat, Energy Balance, and Weight Loss" Anders Gummesson Sahlgrenska Academy University of Gothenburg Sweden 2009.
17. Obesity preventing and managing the global epidemic: Report of WHO consultation on obesity. Geneva, WHO 1997. WHO Tech Rep Ser 2000; 894:I-XII:1- 253.
18. Freedman DS, Khan LK, Serdula MK et al: JAMA 288; 1758, 2002.
19. Montague CT, Farooqi IS, Whitehead JP, et al. Congenital leptin deficiency is associated with severe early-onset obesity in humans. Nature 1997;387:903-908 26.
20. Farooqi S, O'Rahilly S. Genetics of obesity in humans. Endocr Rev 2006;27:710-718.
21. Yeo GS, Farooqi IS, Aminian S, et al. A frameshift mutation in MC4R associated with dominantly inherited human obesity. Nat Genet 1998;20:111-112 28. Vaisse C, Clement K, Guy-Grand B, Froguel P. A frameshift mutation in human MC4R is associated with a dominant form of obesity. Nat Genet 1998;20:113-114 29.
22. Susan sam MD . Obesity and Polycystic Ovary Syndrome Published in final edited form as: Obes Manag. 2007 Apr; 3(2): 69–73 . doi: 10.1089/obe.2007.0019
23. Ansam A. Al-Bayatti .Insulin resistance and upper-body obesity in polycystic ovary syndrome Middle East Fertility Society Journal Vol. No. 3, 2006.
24. Hartz AJ, Barboriak PN, Wong A, et al. The association of obesity with infertility and related menstural abnormalities in women. Int J Obes. 1979;3:57-73.

25. Shilipi pandey, The impact of female obesity on the outcome of fertility treatment journal of human reproductive sciences 2010 May-Aug; 3(2): 62–67.doi: 10.4103/0974-1208.69332

26. Bellver J, Rossal LP, Bosch E, et al. Obesity and the risk of spontaneous abortion after oocyte donation. Fertil Steril.2003; 79:1136-1140.

27. Fedorcsak P, Storeng R, Dale PO, et al. Obesity is a risk factor for early pregnancy loss after IVF or ICSI. Acta Obstet Gyneol Scand: 2000; 79:43-48.

28. Lashen et al Obesity is associated with increased risk of first trimester and recurrent miscarriage: matched case–control study Human Reprod. (2004) 19 (7): 1644-1646. doi: 10.1093/humrep/deh277 First published online: May 13, 2004.

29. Grimble RF. Inflammatory status and insulin resistance. Current Opinion in Clinical Nutrition & Metabolic Care. 2002;5:551–9. [PubMed].

30. Greenberg AS, Obin MS. Obesity and the role of adipose tissue in inflammation and metabolism.American Journal of Clinical Nutrition. 2006;83:461S–465S. [PubMed].

31. Conrad KP, Miles TM, Benyo DF. Circulating levels of immunoreactive cytokines in women with preeclampsia. American Journal of Reproductive Immunology. 1998;40:102–111. [PubMed].

32 Dandona P, Aljada A, Chaudhuri A, Mohanty P, Garg R. Metabolic syndrome: a comprehensive perspective based on interactions between obesity, diabetes, and inflammation. Circulation. 2005;111:1448–54. [PubMed].

33. Sebire NJ, Jolly M, Harris JP, et al: Maternal obesity and pregnancy outcome: A study of 287,213 pregnancies in London. Int J ObesRelatMetabDisord 25:1175, 2001 [PMID: 11477502] .

34. Cedergren MI: Maternal morbid obesity and the risk of adverse pregnancy outcome. Obstet Gynecol. 103:219, 2004 [PMID: 14754687].

35. Weiss JL, Malon FD, Emig D, et al: Obesity, obstetric complications and cesarean delivery rate—a population based screening study. FASTER Research Consortium. Am J ObstetGynecol 190:1091, 2004 [PMID: 15118648]

36. Corie Mcdonald – Wallis ,American journal of obstetrics and gynaecology Volume 209, Issue 3 october 2013, pages 327.el-327.e17.

37. Shin .Y.Kim et al , Percentage of Gestational Diabetes Attributable to Overweight and Obesity, American journal of public health, 2010 June; 100(6): 1047–1052.doi: 10.2105/AJPH.2009.172890.

38. Unterborn J. Pulmonary function testing in obesity, pregnancy, and extremes of body habitus. Clin Chest Med 2001; 22:759±767.

39. Caster LC ,Avina RL Maternal obesity and pregnancy outcome, 2002 Dec;14(6):601-6 DOI:10.1097/01.gco.0000045486.15021.C9

40. Heslehurst, N. et al. (2008) 'The impact of maternal BMI status on pregnancy outcomes with immediate short-term obstetric resource implications: a metaanalysis', Obesity Reviews, 9 (6), pp.635-683.

41. A. Bogaerts, Socio demographic and obstetric correlates of prepregnancy BMI and gestational weight gain, Clinical Obesity © 2013 International Association for the Study of Obesity. clinical obesity 2, 150–159.

42. Crane SS, Wojtwoycz MA, Dye TD, et al. Association between pre-pregnancy obesity and the risk of cesarean delivery. Obestet Gynecol. 1997:89:213-216.

43. Kaiser PS, Kirby RS. Obesity as a risk factor for cesarean in a low risk population. Obestet Gynecol.2001:97:39-43.

44. Sheiner et al: Maternal obesity as an independent risk factor for caesarean delivery; Paediatr Perinat Epidemiol. 2004 May;18(3):196-201 .

45. Dempsey JC et al , Maternal pre-pregnancy overweight status and obesity as risk factors for cesarean delivery. Journal of maternal fetal and neonatal medicine. 2005 Mar;17(3):179-85. DOI:10.1080/14767050500073456.

46. S.Arrowsmith et al, Maternal obesity and labour complications following induction of labour in prolonged pregnancy, BJOG 2011 Apr; 118(5): 578–588.Published online 2011 Jan 26. doi: 10.1111/j.1471-0528.2010.02889.x.

47. Seligman et al : Obesity and gestational weight gain: cesarean delivery and labor complications: Rev. Saúde Pública vol.40 n.3 São Paulo Jun. 2006.

48. Gunilla et al : Trends in caesarean section and instrumental deliveries in relation to Body Mass Index: a clinical survey during 1978 – 2001 Reproductive Health 20107:18 DOI: 10.1186/1742-4755-7-18.

49. Hood DD, Dewan DM, Anesthetic and obstetric outcome in morbidly obese parturients. Anesthesiology. 1993;79:1210-1218.

50. Blomberg M, Maternal obesity and risk of postpartum haemorrhage, Obstet Gynecol. 2011 Sep;118(3):561-8. doi: .1097/AOG.0b013e31822a6c59.

51.Elaine M Fyfe Email author, John MD Thompson, Ngaire H Anderson, Katie M Groom and Lesley M McCowan: Maternal obesity and postpartum haemorrhage after vaginal and caesarean delivery among nulliparous women at term: a retrospective cohort study, BMC Pregnancy and Childbirth 201212:112DOI: 10.1186/1471-2393-12-112.

52. Norman S et al: The effects of degree of obesity on risk for post- caesarean wound complication.Abstract no 157. Am J obstetrics and gynecology 208(1):S78, 2013.

53. Leth et al:Obesity , diabetes and risk of infections diagnosed in hospital and post discharge infections after caesarean section: a prospective cohort study. Acta obstet gynecol scand 90(5):501,2011.

54. Wound infection after cesarean: effect of subcutaneous tissue thickness. Vermillion ST[1], Lamoutte C, Soper DE, Verdeja A.**Obstet Gynecol.** 2000 Jun;95(6Pt1):923-6.

55. Williams Obstetrics 24th Edition.

56. Li R, Jewell S, Grummer-Strawn L: Maternal obesity and breast-feeding practices. Am J ClinNutr 77:931, 2003 [PMID: 12663294] .

57. Jevitt C et al : Lactation complicated by overweight and obesity: supporting the mother and newborn : J Midwifery Womens Health. 2007 Nov-Dec; 52(6):606-13.

58. De Swiet M. Thromboembolic disease. In: James DK, Steer PJ, Weiner CP,Gonik B, editors. High risk pregnancy: management options, 2nd ed.London: W.B. Saunders; 1999. pp. 901±909.

59. LaCoursiere Y, Varner M: The association between prepregnancy obesity and postpartum depression, supported by NIH grant R03-HD-048865. Abstract No. 92. Presented at the 29th Annual Meeting of the Society for Maternal-Fetal Medicine. 26-31 January 2009.

60. Holt VL, Cushing – Haugen KL, Daling J; Body weight and, risk of oral contraceptive failure, Obstet Gynecol 99;820:2002.

61. Cnattingius S, Bergstrom R, Lipworth L, et al, Prepregnancy weight and the risk of adverse pregnancy outcomes. NEJM. 1998; 338:147-152.

62. Flenady V et al :Major risk factor for stillbirth in high outcome countries: A systematic review and meta-analysis. Lancet 377(9774):1331,2011.

63. Waldenstorm U et al :Adverse pregnancy outcome related to advanced maternal age compared with smoking and being overweight. Obstet Gnaecol 123:104,2014.

64. Yao R Ananth C et al :obesity and the risk of still birth : a population cohort study.Am J Obstet Gynaecol 210:s31,2014.

65. Watkins et al: Maternal obesity and risk of birth defects. Paediatrics 111:1152,2003

66. Rasmussen SA, Chu SY et al: Maternal obesity and risk of neural tube defects : a meta-analysis.Am J Obstet and Gynaecol 198(6):611,2008.

67. Gilboa SM, Correa A,et al: Association between pre-pregnancy body mass. index and congenital heart defects. Am J Obstet Gynaecol 202(1):51 el,2010.

68. Ahlsson F., Diderholm B., Jonsson B., Nordén-Lindberg S., Olsson R., Ewald U., Forslund A., Stridsberg M., Gustafsson J. Insulin resistance, a link between maternal overweight and fetal macrosomia in nondiabetic pregnancies. Hormone Research in Paediatrics. 2010;74(4):267–274. doi: 10.1159/000295710.[PubMed]

69. Lindegaard M. L. S., Damm P., Mathiesen E. R., Nielsen L. B. Placental triglyceride accumulation in maternal type 1 diabetes is associated with increased lipase gene expression. Journal of Lipid Research.2006;47(11):2581–2588. doi: 10.1194/jlr.M600236-JLR200. [PubMed] .

70. Catalano P Farrel et al :Long term follow up of infant of women with normal glucose tolerance and GDM: risk factors for obesity and components of metabolic syndrome in child hood. A m J Obstet Gynaecol 193:S3.2005.

71. Schack Neilson et al:High maternal pre-pregnancy weight gain is associated with an increased risk of obesity in childhooh and adulthood independent of maternal BMI. Paediatric res 58:1020,2005.

72. Oken E et al: Maternal weight and gestational weight gain as predictors of long term off-spring growth and health. Presentation at the work shop on the Impact pregnancy weight on maternal and child health, may 30, Washington 2006.

73. James et al.: obesity and the metabolic syndrome, Ann. N.Y. Acad. Sci. 1083: 1–10(2006). C 2006 New York Academy of Sciences. doi: 10.1196/ annals. 1367.002.

74. Carl . J.Lavie et al : obesity and cardiovascular disease : Journal of the American College of Cardiology Vol. 53, No. 21, 2009.

75. Ai Fujimoto : Obesity and Gastrointestinal DiseasesGastroenterology Research and Practice Volume 2013 (2013), Article ID 760574, 6 pages

76. Cogswell ME, Dietz PM, Branum AM: Maternal weight before, during, and after pregnancy in the United States. Presentation at the Workshop on the Impact of Pregnancy Weight on Maternal and Child Health, Washington, DC, May 30, 2006.

77. Alatishe A, Ammori BJ, New JP, et al : Bariatric surgery in women in child bearing age.QJM 106(8):717,2013.

78. Gladys A. Ramos et al. AM J Obstet Gynecol 193:1089-93, 2005.

79. O'Brien TE, Ray JG, Chan WS: Maternal body mass index and the risk of preeclampsia: A systematic overview. Epidemiology 14:368, 2003 , 46.

80. H.M. Salihu : Extreme obesity and risk of placental abruption: Oxford journal, Human Reproduction ,Volume 24, Issue 2 ,Pp. 438-444.

81. Hendler et al :The preterm prediction study:association between maternam BMI and spontaneous and indicated preterm birth. Am J Obstet Gynaecol 2005;192; 882 – 886.

82. Nohr EA et al : prepregnancy obesity andfetal death: a study within the Danish National birth cohort. Obstet Gynaecol 2005;106: 250-259.

83. Chen M et al: Maternal obesity and neonatal Apgar scores:J Matern Fetal Noenatal Med ; 2010 Jan;23(1):89-95.

INFORMATION SHEET

We are conducting a prospective study on **A STUDY ON IMPACT OF MATERNAL OBESITY ON PREGNANCY OUTCOME** in the department of Obstetrics and Gynaecology, Raja Mirasudar Hospital, Thanjavur – 613001.

- At the time of announcing the results and suggestions, name and identity of the patients will be confidential.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator **Signature of participant**

Date:

CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR.SWATHY T.M,** Post Graduate in department of **Obstetrics & Gynaecology, Raja Mirasudar Hospital , Thnjavur 613001**, and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations

Place :

Date : Signature of the Participant

PROFORMA

Serial No: Date of Admission:

Name: Age:

Husbands Name:

Address:

Occupation:

Socioeconomic Status:

Booking:

Immunisation:

History of present illness:

Menstrual history: Regular / Irregular

LMP:

EDD:

Marital History: Married Since:

Consangunity:

Obstetric History: G P L A

Last Child Birth

Previous Obstetric History:

Details of Outcome

Personal History:

Smoking -

Alcohol -

Diet -

Past Medical History:

Diabetes :

Chronic Hypertension :

Heart Disease :

Others :

Drug Intake :

Childhood Obesity :

Past Surgical History:

Present Pregnancy:

I Trimester:

Hyperemisis

Fever

Radiation Exposure

Medications

Pain Abdomen

II Trimester:

Quickening

Bleeding PV

GDM

GHT

Pre-eclampsia

III Trimester:

Bleeding PV

GDM

GHT

Pre eclampsia

GENERAL EXAMINATION

Height at Booking:

Weight at Booking:

BMI at Booking:

Weight at delivery:

Anemia:

Edema:

Pulse: Respiration:

Blood Pressure:

Cardiovascular System:

Respiratory System:

Thyroid:

Breast:

Spine:

Gait:

OBSTETRIC EXAMINATION

Fundal height

Abdominal girth:

Fundal grip:

Umbilical grip:

I pelvic grip:

II Pelvic grip:

Fetal heart:

Liquor volume:

Estimated foetal weight:

PELVIC EXAMINATION:

Investigations:

Urine: Albumin

Sugar

Culture/Sensitivity

Blood:

Hemoglobin:

PCV:

Blood Sugar:

Urea:

S. Creatinine:

Others

Ultra Sound:

ANTEPARTUM COMPLICATION:

Gestational Diabetes:

Pre-eclampsia :

Gestational Hypertension:

Placenta Previa:

Abruptio Placenta:

Malpresentation:

DELIVERY DETAILS:

Induction of Labour: Yes/No

Augmentation: Yes/No

MODE OF DELIVERY:

Labour Natural:

Forceps/ vacuum Delivery:

Caesarean delivery:

Indication for Caesarean delivery:

INTRAPARTUM COMPLICATIONS:

Shoulder dystocia :

Postpartum haemorrhage:

Complete perineal tear:

Colour of the liqour:

Thromboprophylaxis

POSTPARTUM COMPLICATIONS:

Wound Infections:

Wound dehiscence:

Deep vein Thrombosis:

Fever:

NEONATE

Live Born: Still Born: Intrauterine death

Apgar: 1 Min 5 Min

Gestational age at delivery:

Birth weight:

Sex of the baby: M F

Congenital Abnormalities:

Admission in NICU:

Reason for admission in NICU:

Neonatal death:

Duration of Hospital Stay:

Vaginal Delivery:	2 days	>2 days
Cesarean Delivery:	7 days	>7 days

MASTER CHART

S.No	Name	Age	SEC	O-code	HT	WT	BMI	M/H	GDM	GHT	Pre-eclam	ABRUPTION	P.PREVIA	PRETERM	PPROM	TERM	Stillbirth	presentation
1	yasmine nilofer	26	IV	primi	160	95	37.1	REG	_	_	MILD	_	_	_	_	P	_	breech
2	seetha	25	V	primi	164	92	34.2	REG	_	_	MILD	_	_	_	_	P	_	vertex
3	bhuvaneshwari	27	V	G2AI	173	96	32.1	REG	_	_	SEVERE	_	_	_	_	P	_	vertex
4	Vidhya	19	IV	G2AI	154	78	32.9	IR-REG	_	_	MILD	_	_	_	_	P	P	vertex
5	Suganya	22	V	G2A1	149	80	36	REG	_	_	_	_	_	_	_	P	_	vertex
6	Vidhya	25	V	primi	158	79	31.6	REG	_	P	_	_	_	_	_	P	_	vertex
7	Kalaiselvi	33	IV	G3A2	156	80	32.9	REG	_	_	_	_	_	_	_	P	_	vertex
8	Amudha	25	IV	primi	158	82	32.8	REG	INSULIN	_	_	_	_	_	_	P	_	vertex
9	Berlin sophia	26	IV	G2A1	157	81	32.9	REG	_	_	_	_	_	_	_	P	_	vertex
10	Thilagavathy	23	III	primi	168	100	35.4	REG	_	P	_	_	_	_	_	P	_	vertex
11	Arulmathi	19	IV	primi	153	88	37.6	IR-REG	_	_	SEVERE	_	_	_	_	P	_	vertex
12	Amali	28	III	G2P1L1	154	81	34.2	REG	_	P	_	_	_	_	_	P	_	vertex
13	Parameshwari	27	IV	G6P1L1A4	160	84	32.8	REG	_	_	_	_	_	_	P	P	_	vertex
14	Akilandeswari	31	III	G3P2L2	158	84	33.6	REG	_	_	_	_	_	_	_	P	_	vertex
15	Rojapu	26	V	primi	160	82	32	REG	INSULIN	_	MILD	_	_	_	_	P	_	transverse
16	Suganya	28	V	primi	158	84	33.6	IR-REG	_	_	_	_	_	_	_	P	_	vertex
17	Panchavarnam	32	IV	G2P1L1	148	70	32	REG	_	_	_	_	_	_	_	P	_	vertex
18	Susila	20	V	primi	148	85	38.8	REG	_	P	_	_	_	_	_	P	_	vertex

S.No	Name	Age	SEC	O-code	HT	WT	BMI	M/H	GDM	GHT	Pre-eclam	ABRUPTION	P.PREVIA	PRETERM	PPROM	TERM	Stillbirth	presentation
19	Elakiya	22	IV	primi	157	85	34.5	REG	_	_	_	_	_	_	_	P	_	vertex
20	Jeyapriya	24	III	primi	146	65	30.5	REG	_	_	_	_	_	_	_	P	_	vertex
21	Viji vinnarasi	25	II	G2P1L1	157	83	33.7	REG	INSULIN	_	_	_	_	_	_	P	_	vertex
22	Prabhavathy	24	IV	primi	155	94	39.1	IR-REG	_	_	_	_	_	_	_	P	_	vertex
23	Princy	29	V	primi	162	90	34.3	REG	_	P	_	_	_	_	_	P	_	vertex
24	Saranya	25	IV	G2P1L1	164	90	34.3	REG	_	_	_	_	_	_	_	P	_	vertex
25	Nagalaksmi	28	V	G2P1L1	154	83	35.5	REG	MNT	_	_	_	_	_	_	P	_	vertex
26	Malathy	31	IV	primi	152	90	39	REG	_	_	_	_	_	_	_	P	_	vertex
27	Kamala	30	IV	G2P1L1	160	80	31.2	REG	_	P	_	_	_	_	_	P	_	vertex
28	Rajathy	28	V	primi	154	90	37.9	REG	_	_	SEVERE	P	_	_	_	P	P	vertex
29	Kamali	29	III	G3P2L2	160	82	32	REG	_	_	_	_	_	_	_	P	_	vertex
30	Priya	30	IV	G2A1	159	90	35.6	REG	_	P	_	_	_	_	_	P	_	vertex
31	Selvarathi	26	V	primi	160	82	32	REG	INSULIN	_	_	_	_	_	_	P	_	transverse
32	kavitha	21	III	primi	154	89	37.5	REG	_	P	_	_	_	_	_	P	_	vertex
33	Saraswathy	20	V	primi	160	82	32	REG	MNT	_	_	_	_	_	_	P	_	vertex
34	Rani	30	III	G3P1L1A1	162	84	32	REG	_	_	_	_	_	_	_	P	_	vertex
35	Kavyaselvi	26	IV	G2P1L1	159	90	35.6	REG	_	P	_	_	_	_	_	P	_	vertex
36	Priyadharshini	25	V	G3A2	160	86	33.6	REG	_	_	MILD	_	_	_	_	P	_	vertex
37	Shenbagam	22	V	G2A1	163	92	34.6	REG	_	_	_	_	_	_	_	P	_	vertex

S.No	Name	Age	SEC	O-code	HT	WT	BMI	M/H	GDM	GHT	Pre-eclam	ABRUPTION	P.PREVIA	PRETERM	PPROM	TERM	Stillbirth	presentation
38	Taheera banu	20	IV	primi	148	86	39.3	REG	ISULIN	P	_	_	_	_	_	P	_	vertex
39	Padmavathy	26	IV	G4P1L1A2	154	88	37.1	REG	_	_	_	_	_	_	_	P	_	vertex
40	Vidhya	24	IV	G2P1L1	161	89	34.3	REG	_	_	_	_	_	_	_	P	_	vertex
41	Kanimozhli	30	V	primi	166	88	31.9	REG	_	_	SEVERE	_	_	_	_	P	_	vertex
42	Selvi	30	IV	G2P1L0	160	82	32	REG	_	_	_	_	_	_	_	P	_	vertex
43	Nalini	21	III	primi	154	88	37.1	REG	_	_	_	_	_	_	_	P	_	vertex
44	Umadevi	24	IV	G2A1	162	81	30.9	REG	_	P	_	_	_	_	_	P	_	vertex
45	Saritha	22	V	G2P1L1	160	84	32.8	REG	_	_	MILD	_	_	_	_	P	_	vertex
46	Rajathy	24	V	primi	145	68	32.8	REG	_	P	_	_	_	_	_	P	_	vertex
47	Saranya	26	V	G3P2L2	152	79	34.2	REG	INSULIN	_	_	_	_	_	_	P	_	vertex
48	Krishnaveni	28	III	G4P1L1A2	164	82	30.5	REG	_	P	_	_	_	_	_	P	_	vertex
49	Kanmani	22	V	primi	158	79	31.6	IR-REG	_	_	_	_	_	_	_	P	_	vertex
50	kanimozhli	24	V	G3A2	155	80	33.3	IR-REG	_	_	_	_	_	_	P	_	_	vertex
51	Sujatha	28	IV	G3P2L2	153	84	35.9	REG	_	_	_	_	_	_	_	P	_	vertex
52	Riswana parveen	21	IV	primi	154	76	32	REG	MNT	_	_	_	_	_	_	P	_	vertex
53	Christina	22	V	primi	156	74	30.4	REG	_	_	_	_	_	_	_	P	_	vertex
54	Kumari	32	V	G2P1L1	152	82	35.5	REG	_	P	_	_	_	_	_	P	_	vertex
55	Vanitha	28	IV	G2A1	156	78	32.1	REG	_	_	_	_	_	_	_	P	_	vertex
56	Saraswathy	24	V	primi	158	76	30.4	REG	INSULIN	_	_	_	_	_	_	P	_	vertex

S.No	Name	Age	SEC	O-code	HT	WT	BMI	M/H	GDM	GHT	Pre-eclam	ABRUPTION	P.PREVIA	PRETERM	PPROM	TERM	Stillbirth	presentation
57	Kalaiselvi	24	IV	primi	157	81	35.5	IR-REG	_	P	_	_	_	_	_	P	_	vertex
58	Kamatchi	22	IV	G2A1	148	68	31	REG	_	_	_	_	_	_	_	P	_	vertex
59	Parvathy	29	V	G4P2L2A1	154	76	32	REG	_	_	_	_	_	_	_	P	_	vertex
60	Suganya	26	V	G3P2L0	158	79	31.6	REG	_	P	_	_	_	_	_	P	_	vertex
61	Rani	32	III	G2P1L1	159	80	31.6	REG	_	_	MILD	P	_	_	_	P	_	vertex
62	Rajakumari	28	IV	G2A1	162	82	31.2	REG	_	_	_	_	_	_	_	P	_	vertex
63	Kalavathy	32	V	G3P2L2	156	79	32.5	REG	_	_	_	_	_	_	_	P	_	vertex
64	Punitha	20	IV	primi	162	79	30.1	REG	_	_	_	_	_	_	_	P	_	vertex
65	Maheswari	23	III	G2A1	158	76	30.2	REG	INSULIN	P	_	_	_	_	_	P	_	vertex
66	Anitha	24	V	G3P1L1A1	157	80	32.5	REG	_	_	_	_	_	_	_	P	_	vertex
67	Ramya	21	IV	G2P1L0	152	82	35.5	REG	_	P	_	_	_	_	_	P	_	vertex
68	Porkodi	36	V	G4P3L3	150	81	36	REG	_	_	_	_	_	_	_	P	_	vertex
69	Kanimozhli	21	III	G2A1	154	79	33.3	REG	_	P	_	_	_	_	_	P	_	vertex
70	Uma maheswari	20	V	primi	152	88	38.1	IR-REG	_	_	_	_	_	_	_	P	_	vertex
71	Kalaiselvi	26	V	primi	149	68	30.6	REG	_	_	SEVERE	_	_	_	_	P	_	breech
72	kanagavalli	24	III	primi	150	76	33.8	REG	INSULIN	_	_	_	_	_	_	P	_	vertex
73	Thilagavathy	22	V	primi	146	69	32.4	IR-REG	_	P	_	_	_	_	_	P	_	vertex
74	kanmani	24	IV	G2A1	158	82	32.8	REG	_	P	_	_	_	P	_	_	_	vertex
75	Ranjani	26	V	G2P1L1	155	78	32.5	REG	_	_	_	_	_	_	_	P	_	vertex

S.No	Name	Age	SEC	O-code	HT	WT	BMI	M/H	GDM	GHT	Pre-eclam	ABRUPTION	P.PREVIA	PRETERM	PPROM	TERM	Stillbirth	presentation
76	Nithya	28	III	primi	152	85	36.8	IR-REG	MNT	_	_	_	_	_	_	P	_	vertex
77	Kalyani	26	V	G3A2	155	96	40	REG	_	_	_	_	_	_	_	P	_	vertex
78	Rojakannu	24	II	G4A3	154	86	36.3	REG	_	_	_	_	_	_	_	P	_	vertex
79	Pavithra	22	V	G2A1	156	85	34.9	REG	_	_	MILD	_	_	_	_	P	_	vertex
80	Kavitha	21	V	primi	158	82	32.8	REG	_	P	_	_	_	_	_	P	_	vertex
81	Arokiya rani	32	IV	primi	160	90	35.2	REG	_	_	_	_	_	_	_	P	_	vertex
82	Boomadevi	26	III	primi	154	76	32	REG	_	_	_	_	_	_	_	P	_	vertex
83	Anitha	26	V	primi	158	76	30.4	REG	_	P	_	_	_	_	_	P	_	vertex
84	Babitha	30	IV	primi	156	78	32.1	IR-REG	_	_	_	_	_	_	_	P	_	vertex
85	Kanimozhli	28	III	G2P1L1	159	76	30.1	REG	_	_	_	_	_	_	_	P	_	vertex
86	Sugapriya	26	V	G2A1	152	76	32.9	REG	_	_	_	_	_	_	_	P	_	vertex
87	Subashini	27	IV	G3P1L1A1	158	84	33.6	REG	_	P	_	_	_	_	_	P	_	vertex
88	Thilagavathy	26	III	primi	156	82	33.7	REG	_	P	_	_	_	_	_	P	_	vertex
89	Nasreen banu	21	IV	primi	155	80	33.3	IR-REG	_	_	_	_	_	_	_	P	_	vertex
90	Podhumponnu	22	IV	primi	154	79	33.3	REG	_	_	_	_	_	_	_	P	_	vertex
91	Sivaranjani	19	V	G2A1	152	86	37.2	REG	_	P	_	_	_	_	_	P	_	vertex
92	Nandini	20	IV	primi	156	84	34.5	REG	_	_	_	_	_	_	_	P	_	vertex
93	Nadhiya	26	III	primi	160	85	33.2	IR-REG	_	_	_	_	P	_	_	P	_	vertex
94	Kannagi	24	V	G2A1	158	80	32	REG	_	P	_	_	_	_	_	P	_	vertex

S.No	Name	Age	SEC	O-code	HT	WT	BMI	M/H	GDM	GHT	Pre-eclam	ABRUPTION	P.PREVIA	PRETERM	PPROM	TERM	Stillbirth	presentation
95	Sudha	22	IV	G2P1L1	157	82	33.3	REG	_	_	_	_	_	_	_	P	_	vertex
96	Yasodhai	26	IV	G2A1	158	88	35.3	REG	_	_	_	_	_	_	_	P	_	vertex
97	Ambika	28	V	G2P1L1	160	90	35.2	REG	_	_	_	_	_	_	_	P	_	vertex
98	Kalaimani	24	IV	G3P2L1	145	70	33.3	REG	_	_	SEVERE	_	_	_	_	P	_	vertex
99	Tamilselvi	30	V	G4P2L2A1	158	76	30.4	REG	MNT	_	_	_	_	_	_	P	_	vertex
100	Senthamil selvi	21	V	primi	156	88	36.2	REG	_	P	_	_	_	_	_	P	_	vertex

Induction	Mode	Indication	B.WT	M-AS	NICU	PPH	WD INF	DVT	HOS.ST
_	C-S	fpd	3	_	_		P		12
SYN-A	forceps	_	3	_	_				6
GEL	C-S	fail.ind	3.1	P	P				7
SYN-A	LN	_	2.8	_	_				6
	C-S	CPD	2.2	_	_				7
_	C-S	CPD	2.9	_	_				7
_	LN	_	2	_	P				5
SYNTO	LN	_	3.9	_	P				4
_	LN	_	3.2	_	_				3
SYN-A	LN	_	3	_	_				5
GEL/SYN-A	C-S	failto prog	2.6	_	_				10
_	vacuum	_	3.2	_	_				3
SYN-A	forceps	_	2.1	_	P		P		6
SYN-A	LN	_	3.3	_	_				2
_	C-S	TR.LIE	3.9	_	P	P			7
_	C-S	FET DIS	3	_	P				7
SYN-A	LN	_	2.8	_	_				3
SYNTO	LN	_	2.8	_	_				5

Induction	Mode	Indication	B.WT	M-AS	NICU	PPH	WD INF	DVT	HOS.ST
GEL	C-S	fail ind	2.7	_	_				7
SYN-A	LN	_	2.8	_	_				7
GEL/SYN-A	LN	_	3.9	_	P				7
_	C-S	CPD	2.7	_	_				7
_	LN	_	2.1	_	P				5
GEL	C-S	faiilind	3.7	_	_				7
SYN-A	C-S	FAIL PROG	3		P				7
_	C-S	CPD	3.2		_				7
SYN-A	LN		2.9		_				5
SYNTO	LN		2.8		_				6
_	LN		2.1		P				2
_	C-S	CPD	3.9		P				7
_	C-S	TR.LIE	3.9		_		P		16
SYN-A	LN		2.9		_				5
SYNTO	LN		3.8		P				3
SNYTO	LN		3		_				2
_	C-S	CPD	3.2		_				7
GEL/SYN-A	LN		3		_				5
SYN-A	LN		2.9		_				2

Induction	Mode	Indication	B.WT	M-AS	NICU	PPH	WD INF	DVT	HOS.ST
GEL/SYN-A	C-S	FAIL .IND	3		_		P		11
SYN-A	LN		2.9		_				3
_	C-S	CPD	2.8		_				7
SYN-A	C-S	FAIL.PRO	2.7		P				7
_	C-S	FET.DIS	2.7	P	P				7
_	LN		2.8		_				3
SYNTO	C-S	FAIL IND	2.7		_				7
_	LN		2.8		_				5
SYN-A	LN		2.7		_				5
_	C-S	FET .DIS	2.8		_				7
SYN-A	LN		2.6		_				5
GEL/SYN-A	C-S	FAIL.IND	2.7		_				7
SYNTO	C-S	fail.ind	2.3		P				7
SYN-A	LN		3		_				5
GEL/SYN-A	C-S	fail.ind	2.8		_		P		11
SYN-A	C-S	fail .prog	2.7		_				7
_	C-S	CPD	2.7		_				7
SYN-A	vacuum		2.8		_				4
GEL/SYN-A	C-S	FAIL.PROG	3.9		_				7

Induction	Mode	Indication	B.WT	M-AS	NICU	PPH	WD INF	DVT	HOS.ST
GEL	LN		2.7		_				5
SYNTO	LN		3		_				2
_	C-S	CPD	2.8		_				7
SYN-A	LN		3.2		_				5
_	C-S	ABP/UFCX	2.6		_		P		20
SYN-A	vacuum		3.2		_				3
GEL	LN		3		_				5
SYN-A	C-S	FAIL.PROG	3.2		_				7
_	LN		3		_				5
_	C-S	SEVOLIGO	2.5	P	P		P		7
_	LN		3		_				5
_	C-S	CPD	2.1		_				7
SYNTO	LN		2.8		_				5
_	C-S	CPD	2.7		_				7
_	C-S	breech/sp	2.2		_				7
SYN-A	C-S	fail.prog	4		P				7
GEL/SYN-A	C-S	fail.ind	2.7		_				7
_	LN		2.3		_				5
SYN-A	C-S	MSAF	3	P	P		P		12

Induction	Mode	Indication	B.WT	M-AS	NICU	PPH	WD INF	DVT	HOS.ST
_	C-S	FD/OLIGO	2.6		_				7
_	C-S	CPD	3.2		_				7
_	C-S	CPD	2.9		_				7
_	LN		3.8		P				5
_	C-S	CPD	2.8		_				7
SYN-A	forceps		3.5		_	P			3
GEL/SYN-A	C-S	FAIL.IND	2.9		_		P		7
SYNTO	C-S	FAIL.IND	3		_				7
_	LN		2.3		P				7
_	LN		4.1		P				4
SYN-A	C-S	FAIL.PROG	3.8		_				7
_	LN		2.7		_				5
_	LN		2.7		_				5
SYN-A	LN		2.8		_				2
SYNTO	C-S	FAIL.PROG	2.7		_				7
SYN-A	LN		3		_				5
SYN-A	LN		3.8		_				2
_	C-S	P.PR TYIII	4.3		_		P		7
_	C-S	CPD	3.2		_				7

Induction	Mode	Indication	B.WT	M-AS	NICU	PPH	WD INF	DVT	HOS.ST
GEL	LN		3		_				5
SYN-A	LN		3.1		_				5
_	LN		3		_				5
SYNTO	C-S	FAIL.IND	2.6		P				7
_	vacuum		3		_		P		8
_	C-S	SEV.OLIG	2.6		_				7

CONTROL GROUP

NAME	AGE	SEC	0-CODE	HT	WT	BMI	M/H	GDM	GHT	Pre-ecl	Abruption	P.preavia	Preterm	PPROM	TERM	Stillbirth	Presentation	Induction
Kala	22	IV	primi	154	50	21.1	REG	_	_	_	_	_	_	_	P	_	vertex	SYNTO
Sudha	24	V	G2P1L1	156	52	21.4	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Ramya	21	III	primi	158	52	20.8	REG	_	P	_	_	_	_	_	P	_	vertex	_
Anandhi	22	IV	G3A2	160	52	20.3	REG	_	_	_	_	_	_	_	P	_	vertex	_
Prabhavathy	26	V	G2PILI	154	48	20.2	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Seethalakshmi	22	V	G2A1	150	48	21.3	REG	_	_	_	_	_	_	_	P	_	vertex	_
Ambika	23	IV	G2A1	156	52	21.4	IRREG	_	_	MILD	_	_	_	_	P	_	vertex	GEL
Sujatha	24	V	primi	158	52	20.8	REG	_	_	_	_	_	_	_	P	_	vertex	_
Banupriya	22	V	primi	160	56	21.4	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Arthi	25	IV	G2P1L1	154	50	21.1	REG	_	_	_	_	_	_	_	P	_	vertex	_
Suba	27	V	G3P1L1A1	158	52	20.8	REG	_	_	_	_	_	_	_	P	_	vertex	SYNTO
Parvathy	26	V	G3P2L2	156	50	20.5	REG	_	_	_	_	_	_	_	P	_	transverse	_
Vidhya	26	II	primi	145	42	20	REG	_	_	_	_	_	_	_	P	_	vertex	_
Nagalakshmi	23	V	G2P1L1	152	49	21.2	REG	_	_	_	_	_	_	_	P	_	vertex	GEL
Sangeetha	32	IV	primi	154	52	21.9	REG	_	_	_	_	_	_	_	P	_	vertex	_
Renuga	22	V	primi	158	55	22	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Pothumponnu	24	V	primi	160	52	20.3	REG	_	_	_	_	_	_	P	_	_	vertex	_
Subalakshmi	26	IV	primi	162	54	20.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Surya	24	V	G2A1	160	52	20.3	REG	_	_	_	_	_	P	_	_	_	vertex	_
Saranya	24	V	primi	162	57	21.7	REG	_	_	_	_	_	_	_	P	_	vertex	_
Kamali	24	IV	primi	154	48	20.2	REG	__	P	_	_	_	_	_	P	_	vertex	_

NAME	AGE	SEC	0-CODE	HT	WT	BMI	M/H	GDM	GHT	Pre-ecl	Abruption	P.preavia	Preterm	PPROM	TERM	Stillbirth	Presentation	Induction
Pushpa	28	V	primi	156	50	20.2	REG	_	_	_	_	_	_	_	P	_	vertex	_
Rubadevi	24	III	G2P1L1	154	49	20.7	REG	MNT	_	_	_	_	_	_	P	_	vertex	SYNTO
Aparna	27	IV	G2P1L1	155	49	20.4	REG	_	_	_	_	_	_	_	P	_	vertex	_
Sathya	22	V	G2A1	158	50	20	REG	_	_	_	_	_	_	_	P	_	vertex	_
Mahalakshmi	29	IV	G2P1L1	155	52	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Sripriya	22	V	primi	162	55	21	REG	_	_	_	_	_	_	_	P	_	vertex	_
Subashini	30	V	G3P2L2	160	54	21.1	REG	_	_	_	_	_	_	_	P	_	vertex	_
Lakshmi	22	III	G2A1	153	48	20.5	IRREG	_	_	_	_	_	_	_	P	_	vertex	_
Anitha	28	IV	primi	156	50	20.5	REG	_	_	_	_	_	_	_	P	_	vertex	_
karthika	22	V	primi	155	52	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Amirtha	24	V	G2A1	156	53	21.8	REG	_	_	_	_	_	_	_	P	_	vertex	_
Banumathy	23	IV	primi	154	49	20.7	REG	_	P	_	_	_	_	_	P	_	vertex	_
Kannagi	24	V	G2P1L1	157	50	20.3	REG	_	_	_	_	_	_	_	P	_	vertex	_
Shanthi	25	V	G2P1L0	158	51	20.4	REG	_	_	_	_	_	P	_	_	_	vertex	_
Apoorvam	24	III	primi	156	52	21.4	REG	_	_	_	_	_	_	_	P	_	vertex	GEL
Kumari	23	V	G3P1L1A1	159	55	21.8	REG	_	_	_	_	_	_	_	P	_	vertex	_
Padma	22	V	primi	158	52	20.8	REG	_	_	_	_	_	_	_	P	_	vertex	_
Revathy	22	IV	primi	160	56	21.9	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Saraswathy	21	V	G2A1	158	54	21.6	IRREG	_	_	_	_	_	_	_	P	_	vertex	_
Manimegalai	25	V	G2P1L1	153	49	20.9	REG	_	_	_	_	_	_	_	P	_	vertex	_
Kalaiselvi	19	V	primi	154	51	21.5	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Arockiya mary	28	IV	G3P2L2	158	53	21.2	REG	_	_	_	_	_	_	_	P	_	vertex	_
Fathima banu	21	V	G3A2	159	54	21.4	REG	_	_	MILD	_	_	_	_	P	_	vertex	GEL

NAME	AGE	SEC	0-CODE	HT	WT	BMI	M/H	GDM	GHT	Pre-ecl	Abruption	P.preavia	Preterm	PPROM	TERM	Stillbirth	Presentation	Induction
Suryakala	26	V	G2P1L2	161	56	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Kanagavalli	23	V	primi	153	48	20.5	REG	_	_	_	_	_	_	_	P	_	breech	_
Pothumani	22	IV	primi	157	51	20.7	REG	_	_	_	_	_	_	_	P	_	vertex	_
Parvathy	24	V	G2P1L1	156	52	21.4	REG	_	_	_	_	_	_	_	P	_	vertex	_
Vanitha	23	IV	primi	155	52	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Sundari	24	IV	G3P2L2	154	49	20.7	REG	_	_	_	_	_	_	_	P	_	vertex	_
Amutha valli	29	V	G4P2L2A1	158	51	20.4	REG	ISULIN		_	_	_	_	_	P	_	vertex	_
Renuga	24	V	primi	157	51	20.7	REG	_	_	_	_	_	_	_	P	_	vertex	_
Menaga	22	V	primi	159	52	20.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Kanimozhi	30	IV	G3P2L1	162	56	21.3	REG	_	P	_	_	_	_	_	P	_	vertex	_
Ambika	24	IV	G2A1	155	52	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Maheswari	23	V	primi	153	49	20.9	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Kavitha	26	IV	primi	158	50	20	REG	_	_	_	_	_	_	_	P	_	vertex	_
Rani	31	V	G2P1L1	157	51	20.7	REG	_	_	_	P	_	_	_	P	P	vertex	SYN-A
Vidhya	22	V	primi	155	49	20.4	IRREG	_	_	_	_	_	_	_	P	_	vertex	_
Ramya	25	IV	G3P1L1A1	154	49	20.7	REG	_	_	_	_	_	_	_	P	_	vertex	_
Saranya	24	V	G2P1L1	153	50	21.4	REG	_	_	_	_	_	_	_	P	_	vertex	_
Kala	27	V	G3P2L1	156	51	21	REG	_	_	EVER	_	_	_	_	P	_	vertex	SYNTO
Sharmila banu	24	IV	G2P1L1	162	56	21.3	REG	_	_	_	_	_	_	_	P	_	vertex	GEL
Esther	20	V	primi	159	55	21.8	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Selvi	26	V	G2A1	148	45	20.5	REG	_	_	_	_	_	_	_	P	_	breech	_
Sivapriya	27	V	primi	145	46	21.9	REG	_	P	_	_	_	_	_	P	_	vertex	SYN-A
Sathya	24	IV	G2P1L0	154	52	21.9	REG	_	_	_	_	_	_	_	P	_	vertex	_

NAME	AGE	SEC	0-CODE	HT	WT	BMI	M/H	GDM	GHT	Pre-ecl	Abruption	P.preavia	Preterm	PPROM	TERM	Stillbirth	Presentation	Induction
Anitha	26	IV	G2P1L1	157	50	20.3	REG	_	_	_	_	_	_	_	P	_	vertex	_
Annamayil	22	V	G3P2L2	153	51	21.8	REG	_	_	_	_	_	_	_	P	_	vertex	_
Vaidehi	32	V	primi	155	50	20.8	REG	_	_	_	_	_	_	_	P	_	vertex	_
Jeyamani	24	V	G4P1L1A2	158	54	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Kamala	25	IV	G2P1L1	161	57	22	REG	_	_	_	_	_	_	_	P	_	vertex	_
Jeyanthi	24	V	primi	158	54	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Mahalakshmi	26	IV	primi	157	54	21.9	REG	_	_	_	_	_	_	_	P	_	vertex	_
Sripriya	24	IV	primi	158	51	20.4	REG	_	_	_	_	_	_	_	P	_	vertex	_
Malliga	23	V	G2A1	155	49	20.4	REG	_	_	_	_	_	_	_	P	_	vertex	_
Lakshmi	25	V	G2P1L1	156	52	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Victoria	24	V	G3P2L0	149	46	20.7	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Kalaimani	30	V	G2P1L1	156	51	21	REG	_	_	_	_	_	_	_	P	_	vertex	_
Parvathy	26	III	G2P1L1	155	52	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Thangam	23	IV	G2A1	162	57	21.7	REG	_	_	_	_	_	_	_	P	_	vertex	_
Ramya	22	V	primi	157	51	20.7	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A
Revathy	24	V	primi	157	53	21.5	REG	_	_	_	_	_	_	_	P	_	vertex	_
Amsavalli	23	V	G3P2L2	159	54	21.4	REG	ISUL	_	_	_	_	_	_	P	_	vertex	_
Karpagam	25	V	G2P1L1	157	52	21.1	REG	_	_	_	_	_	_	_	P	_	vertex	_
Marikannu	24	IV	G4P3L2	153	49	20.9	REG	_	_	_	_	_	_	_	P	_	vertex	SYNTO
Vimala	26	V	G3A2	154	50	21.1	REG	_	_	_	_	P	_	_	P	_	vertex	_
Yasmine Banu	22	V	primi	162	57	21.7	IRREG	_	_	_	_	_	_	_	P	_	vertex	_
Anandhi	23	V	primi	155	51	21.2	REG	_	_	_	_	_	_	_	P	_	vertex	_
Suganya	26	IV	G2P1L1	156	49	20.1	REG	_	_	_	_	_	_	_	P	_	vertex	SYN-A

NAME	AGE	SEC	0-CODE	HT	WT	BMI	M/H	GDM	GHT	Pre-ecl	Abruption	P.preavia	Preterm	PPROM	TERM	Stillbirth	Presentation	Induction
Kamatchi	27	V	G2P1L1	158	52	20.8	REG	_	_	_	_	_	_	_	P	_	vertex	_
Gayathri	22	III	primi	148	45	20.5	REG	_	_	_	_	_	_	_	P	_	vertex	_
Kalaivani	23	V	G2A1	150	48	21.3	REG	_	_	_	_	_	_	P	_	_	vertex	SYNTO
Sumithra	24	V	primi	155	51	21.2	REG	_	_	_	_	_	_	_	P	_	vertex	_
Jeyakodi	26	IV	primi	163	58	21.8	REG	_	_	_	_	_	_	p	_	_	vertex	_
Priya	23	V	G2P1L0	159	55	21.8	REG	_	_	_	_	_	_	_	P	_	vertex	_
Santhiya	28	V	primi	157	52	21.1	REG	_	_	_	_	_	_	_	P	_	vertex	_
Amaravathy	24	IV	G2P1L1	158	54	21.6	REG	_	_	_	_	_	_	_	P	_	vertex	_
Tamilselvi	27	IV	G2A1	156	51	21	REG	_	_	_	_	_	_	_	P	_	vertex	_
Renuga	28	V	primi	160	56	21.9	REG	_	_	_	_	_	_	_	P	_	vertex	_

NAME	MODE	IND	B.WT	MEC-AS	NICU	PPH	RET.PL	WND-INF	DVT	HOSP-ST
Kala	LN	_	2.6	_	_	_	_	_	_	2
Sudha	LN	_	2.6	_	_	_	_	_	_	2
Ramya	LN	_	2.8	_	_	_	_	_	_	5
Anandhi	C-S	CPD	3	_	_	_	_	_	_	7
Prabhavathy	LN	_	3.1	_	_	_	_	_	_	2
Seethalakshmi	LN	_	2.8	_	_	_	_	_	_	2
Ambika	VACUUM	_	2.9	_	_	_	_	_	_	5
Sujatha	LN	_	2.8	_	_	_	_	_	_	2
Banupriya	LN	_	2.7	_	_	_	_	_	_	2
Arthi	LN	_	2.7	_	_	_	_	_	_	2
Suba	LN	_	3.1	_	P	P	_	_	_	3
Parvathy	C-S	tran. Lie	2.9	_	_	_	_	_	_	7
Vidhya	LN	_	2.7	_	_	_	_	_	_	2
Nagalakshmi	C-S	FAIL.IND	2.9	_	_	_	_	_	_	7
Sangeetha	LN	_	3	_	_	_	_	_	_	2
Renuga	LN	_	3.1	_	_	_	_	_	_	2
Pothumponnu	LN	_	2	_	P	_	_	_	_	4
Subalakshmi	LN	_	2.9	_	_	_	_	_	_	2
Surya	LN	_	2.1	_	_	_	_	_	_	2
Saranya	LN	_	2.2	_	_	_	_	_	_	2
Kamali	LN	_	2.8	_	_	_	_	_	_	5

NAME	MODE	IND	B.WT	MEC-AS	NICU	PPH	RET.PL	WND-INF	DVT	HOSP-ST
Pushpa	LN	_	3.2	_	_	_	_	_	_	2
Rubadevi	LN	_	3	_	P	_	_	_	_	2
Aparna	LN	_	3.2	_	_	_	_	_	_	2
Sathya	LN	_	3.1	_	_	_	P	_	_	3
Mahalakshmi	LN	_	3	_	_	_	_	_	_	2
Sripriya	LN	_	2.8	_	_	_	_	_	_	2
Subashini	LN	_	2.8	_	_	_	_	_	_	2
Lakshmi	C-S	CPD	3.2	_	_	_	_	_	_	7
Anitha	LN	_	2.9	_	_	_	_	_	_	2
karthika	LN	_	2.7	_	_	_	_	_	_	2
Amirtha	LN	_	2.8	_	_	_	_	_	_	2
Banumathy	LN	_	2.8	_	_	_	_	_	_	5
Kannagi	LN	_	2.7	_	_	_	_	_	_	2
Shanthi	LN	_	1.8	_	P	_	_	_	_	5
Apoorvam	LN	_	2.8	_	_	_	_	_	_	2
Kumari	LN	_	2.9	_	_	_	_	_	_	2
Padma	C-S	FET.DIS	2.8	_	P	_	_	_	_	7
Revathy	LN	_	2.7	_	_	_	_	_	_	2
Saraswathy	FORCEPS	_	3.1	_	_	_	_	_	_	3
Manimegalai	LN	_	2.9	_	_	_	_	_	_	2
Kalaiselvi	LN	_	2.9	_	_	_	_	_	_	2
Arockiya mary	LN	_	3	_	_	_	_	_	_	2
Fathima banu	FORCEPS	_	2.8	_	_	_	_	P	_	7

NAME	MODE	IND	B.WT	MEC-AS	NICU	PPH	RET.PL	WND-INF	DVT	HOSP-ST
Suryakala	LN	_	2.7	_	_	_	_	_	_	2
Kanagavalli	C-S	FPD	2.8	_	_	_	_	_	_	7
Pothumani	LN	_	2.9	_	_	_	_	_	_	2
Parvathy	LN	_	2.8	_	_	_	_	_	_	2
Vanitha	LN	_	3.1	_	_	_	_	_	_	2
Sundari	LN	_	3	_	_	_	_	_	_	2
Amutha valli	LN	_	3.7	_	P	_	_	_	_	3
Renuga	LN	_	3.2	_	_	_	_	_	_	2
Menaga	LN	_	2.9	_	_	_	_	_	_	2
Kanimozhi	LN	_	2.8	_	_	_	_	_	_	5
Ambika	LN	_	2.9	_	_	_	_	_	_	2
Maheswari	LN	_	2.8	_	_	_	_	_	_	2
Kavitha	LN	_	2.7	_	_	_	_	_	_	2
Rani	LN	_	2.6	_	_	_	_	_	_	5
Vidhya	LN	_	3	_	_	_	_	_	_	2
Ramya	LN	_	3.1	_	_	_	_	_	_	2
Saranya	LN	_	3.1	_	_	_	_	_	_	2
Kala	C-S	FAIL.IND	2.5	_	_	_	_	_	_	7
Sharmila banu	LN	_	2.8	_	_	_	_	_	_	2
Esther	LN	_	2.9	_	_	_	_	_	_	2
Selvi	C-S	FPD	3.2	_	_	_	_	_	_	7
Sivapriya	LN	_	2.5	_	_	_	_	_	_	5
Sathya	LN	_	3.2	_	_	_	_	_	_	2

NAME	MODE	IND	B.WT	MEC-AS	NICU	PPH	RET.PL	WND-INF	DVT	HOSP-ST
Anitha	LN	_	3	_	_	_	_	_	_	2
Annamayil	LN	_	2.9	_	_	_	_	_	_	2
Vaidehi	LN	_	2.8	_	_	_	_	_	_	2
Jeyamani	LN	_	2.7	_	_	_	_	_	_	5
Kamala	LN	_	3	_	_	_	_	_	_	2
Jeyanthi	LN	_	2.9	_	_	_	_	_	_	2
Mahalakshmi	LN	_	2.9	_	_	_	_	_	_	2
Sripriya	LN	_	2.8	_	_	_	_	_	_	2
Malliga	LN	_	2.8	_	_	_	_	_	_	2
Lakshmi	LN	_	2.6	_	_	_	_	_	_	2
Victoria	LN	_	2.8	_	_	_	_	_	_	2
Kalaimani	LN	_	3.3	_	_	_	_	_	_	2
Parvathy	LN	_	3.2	_	_	_	_	_	_	2
Thangam	LN	_	3.1	_	_	_	_	_	_	2
Ramya	LN	_	3.1	_	_	_	_	_	_	2
Revathy	LN	_	2.6	_	_	_	_	_	_	2
Amsavalli	LN	_	3.4	_	P	_	_	_	_	2
Karpagam	LN	_	2.8	_	_	_	_	_	_	2
Marikannu	LN	_	2.9	_	_	_	_	_	_	2
Vimala	C-S	TYPE III	2.8	_	_	_	_	_	_	7
Yasmine Banu	LN	_	2.5	_	_	_	_	_	_	2
Anandhi	LN	_	3.2	_	_	_	_	_	_	2
Suganya	LN	_	3.1	_	_	_	_	_	_	2

NAME	MODE	IND	B.WT	MEC-AS	NICU	PPH	RET.PL	WND-INF	DVT	HOSP-ST
Kamatchi	LN	_	2.8	_	_	_	_	_	_	2
Gayathri	C-S	FET.DIS	2.9	_	_	_	_	_	_	7
Kalaivani	LN	_	2.1	_	_	_	_	_	_	4
Sumithra	LN	_	3	_	_	_	_	_	_	2
Jeyakodi	LN	_	2.9	_	_	_	_	_	_	2
Priya	LN	_	2.6	_	_	_	_	_	_	2
Santhiya	LN	_	2.7	_	__	_	_	_	_	5
Amaravathy	LN	_	2.8	_	_	_	_	_	_	2
Tamilselvi	LN	_	2.8	_	_	_	_	_	_	2
Renuga	LN	_	2.2	_	_	_	_	_	_	2

KEY TO MASTER CHART

HT- Height

Wt – Weight

SEC – socioeconomic class

BMI- body mass index

GDM- gestational diabetes

GHT- gestational hypertension

PPROM – preterm premature rupture of membranes

P.previa – placenta previa

C-S- Caesarean

LN- labour natural

Fail .ind – failed induction

Fet .dis- fetal distress

MSAF- meconium stained amniotic fluid

Fail . prog – failure to progress

NICU- neonatal intensive care unit

HOS.ST – hospital stay

PPH- postpartum haemorrhage

DVT- Deep vein thrombosis

WD INF- wound infection